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The Journal of
Bone and Joint Surgery

EDITORIALS AND ANNOTATIONS

SOME PROBLEMS OF METALS AND THEIR TRANSFER IN ORTHOPAEDIC SURGERY

The implantation in the body of heterogenous materials is being practised on an ever increasing scale. Some of the results are encouraging, but the number of failures indicates that many of the problems associated with these procedures are not yet clearly understood. Many materials are now available which appear, at first sight, suitable for orthopaedic purposes. However, reconstruction and replacement of portions of the human skeleton pose a multitude of biological and mechanical problems, not the least of which is the need for a more comprehensive understanding than we have, at present, of the basic responses of the cell to immediate and remote physico-chemical influences.

Various investigations into the behaviour of metals *in vivo* and *in vitro* [1-7] have caused considerable confusion in the appreciation of their relative inertness, and in many instances the conjoint use of apparently similar but in fact dissimilar metals has obscured the behaviour of the individual metal. Bowden, Williamson and Laing have drawn attention to this problem by work which is reported in this issue of the Journal. They point out that when two metals in contact are subject to relative displacement under load, transfer of one material to the other occurs; further, they postulate that some failures can be explained in terms of electro-chemical phenomena which may take place between the implant and the deposited particles of metal. While from their observations there is no doubt that their deductions are correct, they may only be a part of the whole picture: there is sufficient evidence to suggest that local plastic deformation of an implant may also contribute towards failure. Their tests were relatively brief, and the authors might have based a part of their conclusions on the effects of stress corrosion. While materials in an unstressed state may show no sign of corrosion, they may behave very differently when in a stressed condition. The term "stress corrosion" implies that the material has been subject to the simultaneous action of static stress and the corrosive medium, resulting in more rapid deterioration of its mechanical properties than would take place by the separate but additive action of these two factors. Metallurgical studies (Edeleanu 1953) [8] show that strained austenitic steels are seldom single-phase but rather contain a martensitic phase (or pseudo-martensite) formed during straining of metastable austenite. It is believed that in stainless steels this pseudo-martensite, formed on straining, is the source of transgranular stress corrosion. Bowden *et al.* have found quantitatively in certain areas that the amount of metallic ions present in the surrounding tissue considerably exceeded that which is usually transferred to the screw head and thus deduced that the bulk of the implant was subject to corrosion. Despite the authors' explanation that the deposited fragments of metal from tools might have contributed to the initiation of corrosion of the implant, the problem of plastic deformation of metal and its effect on corrosion resistance should not be overlooked. Considerable torque is exerted by the operator when a screw is inserted into bone. For a given torque, the amount of damage to the head of the screw will be a function of the relative size of the screw-driver tip and the screw slot. Local high intensity deformation will occur in the slot of the screw if a thin loosely fitting screw-driver is used. Only a screw-driver with a tip which accurately fits the slot in the screw will do the least amount of damage. Once deformation of the structure of the steel of the screw has occurred, corrosion will be initiated, the transferred metal from the screw-driver probably accelerating the corrosion.

We believe that stainless steel compound implants—that is implants consisting of several components, for example plates and screws—will always be liable to failure. For instance, it has

been found that at the contact point between stainless steel screws and plates, a type of fretting corrosion has occurred. This fretting corrosion has resulted because at the plane of contact, between the screw and the plate, a small slip has taken place from cyclic stressing of the implant before union of the fracture. This fretting corrosion can be accelerated in the presence of transferred metal from, for instance, screw-holding forceps made of dissimilar metal.

Besides the problem of transfer of metals to the implant, there is that associated with the use of power-operated tools, where sharp and therefore relatively thin and brittle edges of metal are brought into contact with the bone. As both Bowden *et al.* and Shaw and Scales (1954) [9] have found, there can be a deposition of appreciable amounts of metals on the bone. The physical state and composition of this transferred metal will be different from that of the metal implant which may be placed in proximity to these contaminated areas. Further, tissue fluid is an electrolyte and thus two electrodes will be formed with consequent release of ions. It is not possible to say how these ions may influence metabolic processes, but it is well known that iron and chromium can react with the protein molecule and it is conceivable that in this manner enzyme-initiated reactions can be prevented. In the vicinity of corrosion—that is the destruction of metal by chemical or electro-chemical action—calcification is always inhibited, the area being walled off by fibrous tissue.

Many surgeons prefer to use stainless steel screws because they find that it is easier to remove them subsequently as the bone does not form in apposition to the thread. This is in contrast to the cobalt-chrome alloy screws which are usually extremely difficult to extract. This would suggest that the products of micro-corrosion cause stimulation of the fibroblast rather than the osteoblast. In the cutting of bone grafts, the transfer of metals may also be a factor which may influence the result of the procedure, particularly when power-operated tools with new saw blades are employed. From experiments we have found that at 2,200 revolutions a minute the transfer of metal is three times as great as when the same tool is used for the same purpose at 700 revolutions a minute. With carbide-tipped tools, however, the transfer of metal over the same wide range of speeds is less and constant.

Bowden, Williamson and Laing's work again emphasises the problems associated with the use of stainless steel. The fact that an implant is made from austenitic stainless steel means little. Its corrosion resistance depends on a variety of factors, many of them beyond the control of either the manufacturer or the surgeon. It is obvious that the transfer of metals is important; its true significance, however, can only be established after further complicated experimental work. We believe that the cobalt-chrome alloys have in clinical experience proved to be so superior in all respects to the existing stainless steels that they should now be very seriously considered for all surgical implants. Further, it is possible to tip all instruments with the alloy so that the problem of the transfer of dissimilar metals may not result. We have never seen a cobalt-chrome implant which has failed either mechanically or biologically because of corrosion phenomena. While this change-over would involve considerable expenditure, the net saving to the community would be considerably greater. JOHN T. SCALES and J. M. ZAREK.

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THE FANCONI SYNDROME

Metabolic Studies on Treatment

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LILY MULLIGAN and MARGARET CAREY, LONDON, ENGLAND

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The association of rickets or osteomalacia with a variety of renal tubular defects, probably congenital, has been called by many different names such as resistant rickets, Fanconi syndrome, and hyperchloraemic acidosis with nephrocalcinosis. They have been classified by Dent (1952) into six main types according to the various combinations of recognisable defects of tubule function. The Fanconi syndrome (Dent Type 4), with which this paper is alone concerned, is characterised by rickets or osteomalacia with an associated deficiency of tubular reabsorption of phosphate, glucose, certain amino acids and sometimes of water and potassium. The inability of the kidney to maintain the acid-base balance of the body causes a chronic acidosis and loss of calcium. Although the main lesion is tubular, there is frequent evidence of glomerular damage as shown by an impaired glomerular filtration rate and presence of proteinuria.

In the commonest form of this syndrome symptoms begin in early life, the child being brought for advice at one to two years of age with active rickets which fails to respond to normal doses of vitamin D. Other symptoms related to the associated electrolyte defect may also be present. Occasionally the defect does not become apparent until middle life, when osteomalacia is found, perhaps with pseudo-fractures. We are reporting a case from each of these groups.

There are many accounts of this interesting and rare syndrome. The literature between 1926 and 1943 was reviewed by McCune, Mason and Clarke (1943) when reporting a case of their own. Stowers and Dent (1947) published detailed observations and necropsy findings on an adult patient. Cooke *et al.* (1947) also reported an adult case, as did Milne, Stanbury and Thomson (1952). Linder, Bull and Grayce (1949) investigated a case associated with retinitis pigmentosa.

Treatment described in the earlier literature has often been unsatisfactory. In more than half the cases, both in adults and children, the patients were dead when reported. Great improvement, both radiological and symptomatic, has been reported recently when therapy, more carefully aimed at correcting the electrolyte changes, has been applied (Bickel and Smellie 1952, Sirota and Hamerman 1954, Dragsted and Hjorth 1953, Dent and Harris 1951). No patient thus treated has yet been followed for more than a few years; so the long term prognosis remains obscure.

Recent principles of treatment have involved the administration of sodium bicarbonate, calcium, vitamin D, potassium and phosphate.

In this paper balance studies on two cases of the Fanconi syndrome are presented to show the effects upon calcium and phosphorus metabolism of combining correction of the acidosis with high dosage of vitamin D.

CASE REPORTS

Case 1—A boy, thirteen years old at the time of this investigation, had presented with severe rickets at fifteen months. Polyuria and polydipsia had been noted since infancy, and deformities of the legs later required osteotomies. Investigations had repeatedly shown a

TABLE I

CASE I—SUMMARY OF INVESTIGATIONS WITH RESULTS

Blood	
Serum calcium	10·2 mg./100 ml.
Serum inorganic phosphorus	2·8 mg./100 ml.
Alkaline phosphatase	57·3 King-Armstrong units
Alkali reserve	22·3 m. eq/l.
Potassium	4·3 m. eq/l.
Sodium	141·0 m. eq/l.
Albumin	5·58 g./100 ml.
Globulin	2·21 g./100 ml.
Total plasma protein	7·79 g./100 ml.
Renal tract	
Blood urea	20·7 mg./100 ml.
Urea clearance	92·5 per cent in first hour. Average normal 124·0 per cent in second hour. Average normal
Glucose tolerance test	Normal (glucose present in all urine specimens)
Urine	
Albumin	Nil
Glucose	+(intermittently)
Cytology	Normal
Ammonia	0·62 g. (as N per day)
pH	6·4
Fat balance	
On an intake of	75·3 g. per day
Absorption	93·0 per cent of ingested fat over six days
Radiographs	
Typical appearances of active rickets	
Paper chromatography	Gross amino-aciduria on many occasions
Slit lamp examination of cornea showed no cysteine crystals	

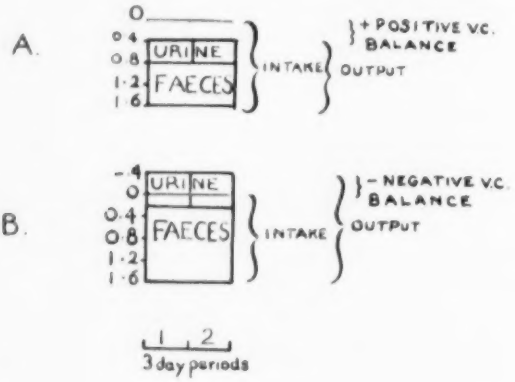

FIG. 1

In these metabolic charts the intakes are measured vertically downwards from the baseline (*red*) marked O. Positive balance is indicated by the fact that the rectangles showing urinary and faecal output fall below this baseline—that is, excretion is less than intake (A) and negative balance when they reach above the baseline—that is, excretion is greater than intake (B).

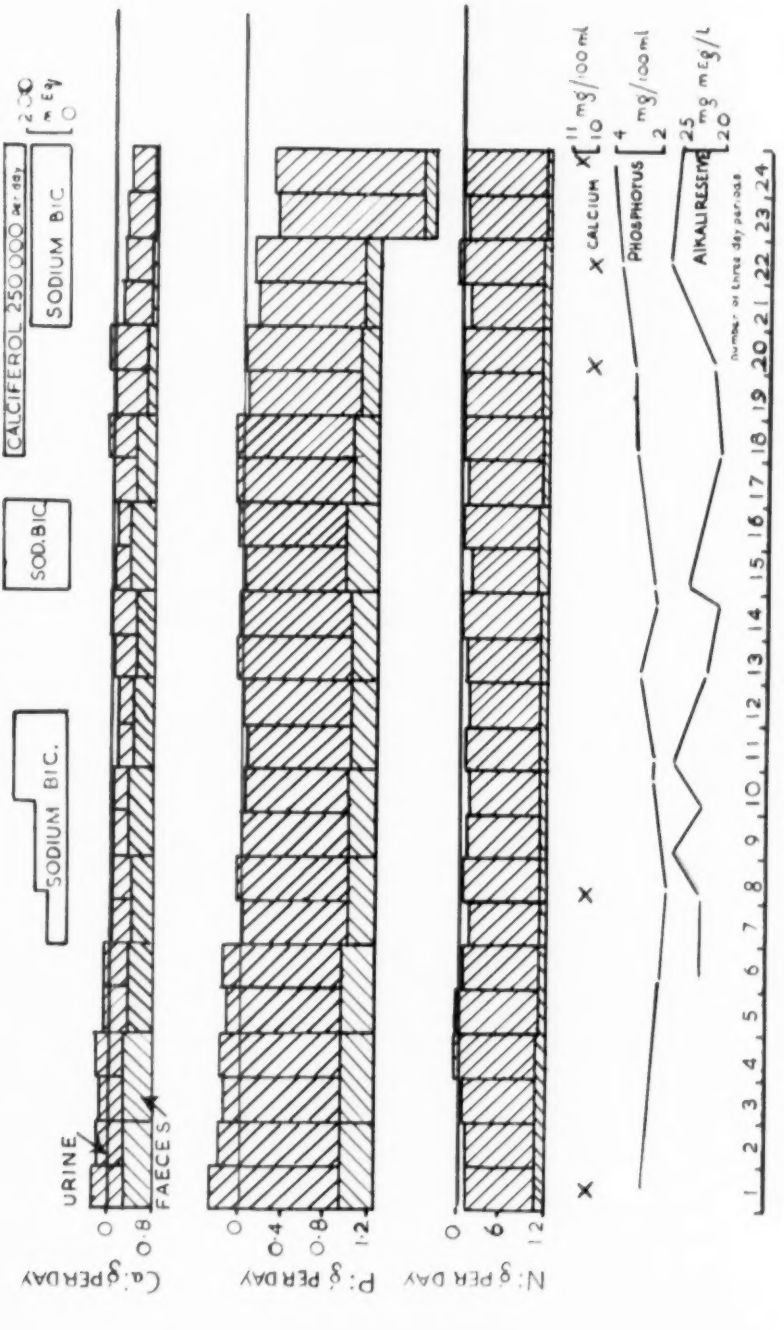

FIG. 2

Case 1—Balance data for calcium, phosphorus and nitrogen for twenty-four consecutive three-day periods. Note: 1) The very high urine calcium and phosphorus put patient into negative balance for calcium and phosphorus in the control periods 1 to 6. 2) Increasing doses of sodium bicarbonate in periods 7 to 12 caused reduction in urine calcium and phosphorus. Faecal calcium unaffected; patient in balance for calcium and phosphorus. 3) Increase in urine calcium on stopping sodium bicarbonate with further lowering of urine calcium when it was recommended in periods 15 to 16. Stopping the alkali in period 17 once again caused a rise in urine calcium. 4) In periods 18 to 24 combined alkalies and calciferol put the patient into positive calcium and phosphorus balance.

low serum inorganic phosphorus, a high alkaline phosphatase, chronic acidosis, glycosuria and gross amino-aciduria. For six years he was treated with 2·5 milligrams (100,000 units) daily of calciferol, with considerable radiographic improvement, but he remained dwarfed and had several hypercalcaemic episodes. The latter occurred when the dose of vitamin D was increased in an attempt to raise his serum phosphate to normal childhood levels. The serum inorganic phosphorus on non-toxic dosage of vitamin D (2·5 milligrams daily) rose from 1·5 to 3·5 milligrams/100 millilitres and alkaline phosphatase fell from 160 units to

TABLE II

CASE 2—SUMMARY OF INVESTIGATIONS, WITH RESULTS

Blood	
Haemoglobin	100 per cent (14·8 g.)
Erythrocyte sedimentation rate	1 mm. (Wintrobe)
Leucocytes	Normal
Serum calcium	10·2 mg./100 ml.
Phosphorus	1·2 mg./100 ml.
Alkaline phosphatase	15 King-Armstrong Units
Alkali reserve	17 m. eq/l.
Albumin	4·0 g./100 ml.
Globulin	2·5 g./100 ml.
Total serum protein	6·5 g./100 ml.
Electrophoretic pattern	Normal
Renal tract	
Blood urea	37 mg./100 ml.
Urea clearance	42 per cent in the first hour
Glucose tolerance test	Normal; glycosuria at all levels of blood sugar
Urine	
Albumin	300 mg. per cent
Glucose	0·1 per cent
Amino acids	Very great excess—in the neighbourhood of 9 g. per day. (Two-way partition paper chromatography)
Cytology	Normal
Culture	Sterile
Ammonia	0·42 g. of ammonia N/24 hours
Fat balance	
On an intake	50 g. per day
Absorption	91 per cent over a period of ten days

Paper chromatography

A heavy amino aciduria was observed when the usual fraction of the twenty-four hour urine was run. Amino acids present in this urine, and which are not normally observed, were: proline, lysine, arginine, cysteine, and two spots in the leucine-valine position. Figures for the excretion of glutamine, alanine glycine, threonine, and proline, obtained by scaling down the fraction of urine run on the chromatogram, were as follows (figures in moles $\times 10^{-5}$ day): glutamine 2,370 (normal 80); alanine 1,420 (normal 40); glycine 950 (normal 120); threonine 470 (normal 40–120); proline 560 (normal nil).

around 35 units, but hypercalcaemia preceded the full attainment of normal figures, and his polyuria greatly increased (Stevenson 1950).

Investigations—The relevant investigations are summarised in Table I.

Metabolic balance studies—The balance data are charted in Figure 2.

Case 2—A man of forty-seven years, a stoker, had for two years complained of " rheumatic " pains in his feet, left hip and chest; these and difficulty in walking had caused him to stop work. He was found to have pseudo-fractures of the neck of the left femur and the right

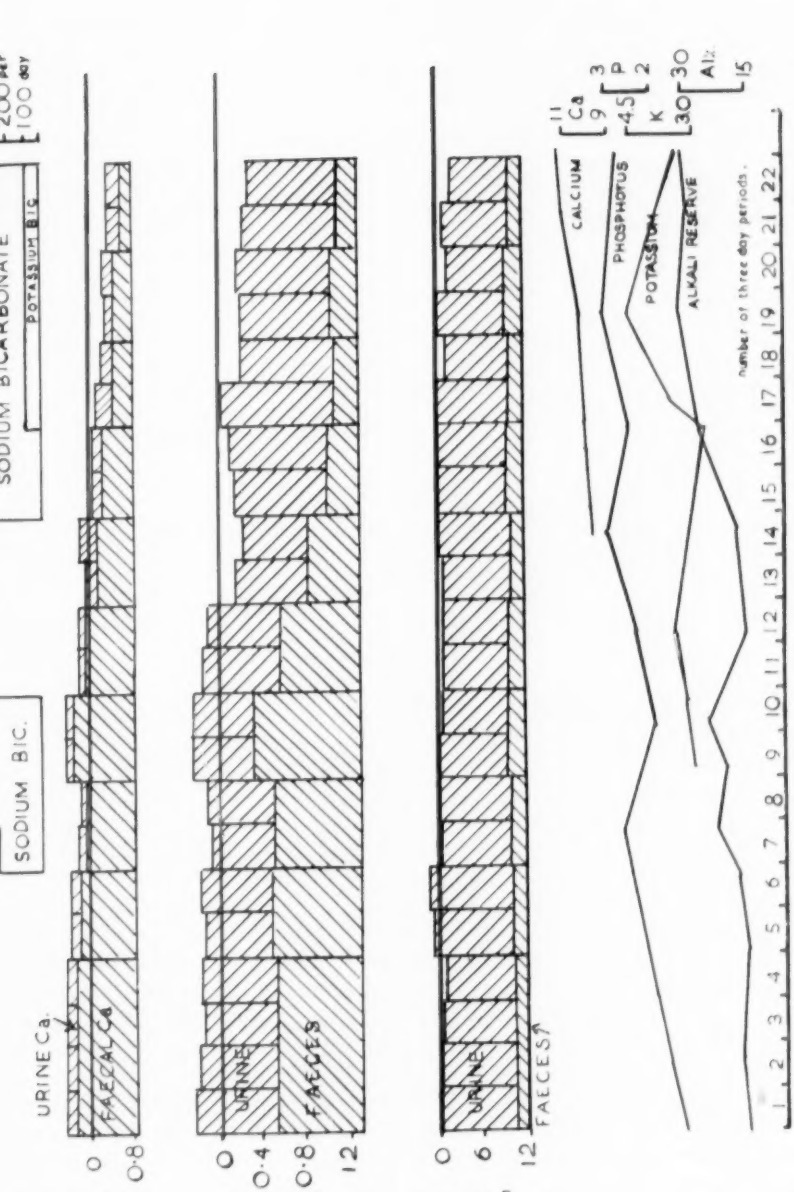

Fig. 3

Case 2—Balance data for calcium, phosphorus and nitrogen for twenty-two consecutive three-day periods. Note: 1) The negative calcium and phosphorus balance in control periods 1 to 6 due to faecal calcium being greater than the intake and causing a pathologically high proportion of faecal phosphorus. 2) In periods 7 to 10 increasing doses of sodium bicarbonate lowered urine calcium from 200 milligrams to 70 milligrams daily. No obvious effect on faecal calcium or phosphorus. 3) When the sodium bicarbonate was discontinued the urine calcium rose. 4) Calciferol in periods 13 to 14 caused a further rise in urine calcium, as well as lowering faecal calcium. Balance unaltered. 5) Finally combined alkali and calciferol resulted in strongly positive balance for calcium, phosphorus and nitrogen.

pubic ramus and bilateral multiple fractures of ribs; none showed any sign of healing. He had a low serum inorganic phosphorus, normal calcium, low potassium, raised alkaline phosphatase and moderate acidosis. His urine contained albumin, glucose and animo acids; the last to the extent of 9 grammes per twenty-four hours as roughly measured by paper chromatography.

Investigations—The relevant investigations are summarised in Table II.

Metabolic balance studies—The balance data are charted in Figure 3.

METHODS

The patients were admitted to the Metabolism Unit. Neither was confined to bed. The metabolic régime and the collection of excreta followed the methods advocated by Albright and Reifenstein (1948). Urine was collected in three-day periods and faeces in six-day periods. Faecal collections were demarcated by the administration of 0·5 grammes of carmine. The constancy of faecal nitrogen was used as a check on the accuracy of the collections.

Food was prepared by the dietitian in the Metabolic Unit kitchen. Bulk supplies of meat, biscuits and homogenised milk were used. Bread was made in the Unit from unfortified flour. Distilled water was used throughout. The diets were analysed twice during the balance. The patients were on diet for at least one week before balance studies were recorded.

Throughout the studies, calcium in the serum, food, faeces and urine was estimated by the method of Clark and Collip (1925), phosphorus by the method of Briggs (1922), and nitrogen by the method of Koch and McMeekin (1924). The serum inorganic phosphorus and alkaline phosphatase was estimated by the method of King (1951). Faecal fat was estimated by a modification of King's method (1951).

Serum potassium was estimated by the method of King (1951), chlorides by the method of Van Slyke (1923), and blood sugar by the method of King and Garner (1947). Blood urea was estimated by the method of King (1951) and urine urea by MacLean's ureometer.

Plasma CO^2 combining power was estimated by the method of Van Slyke and Cullen (1917).

For urine ammonia estimation our specimens were stored in the refrigerator and toluene was used as a preservative. The estimations were carried out as soon as the entire specimen had been collected.

DISCUSSION

Bone disease in the Fanconi syndrome results from two distinct mechanisms. Fanconi was the first to suggest that there was a renal tubular defect with consequent loss of phosphate, sugar and amino acids. As a result of the lowered renal threshold for phosphate there is a chronic hypophosphatemia; consequently the ionic product of calcium x phosphorus is so low that the calcium-phosphate complex fails to be precipitated into osteoid. In this way osteomalacia or rickets results as it does in the related condition resistant rickets. This defect, as in resistant rickets, can be treated with massive doses of vitamin D.

The second defect in the Fanconi syndrome results in chronic acidosis. The nature of this lesion is debatable. Latner and Burnard (1950) claimed to have shown that the defect responsible for acidosis in hyperchloraemic acidosis was a failure of bicarbonate reabsorption in the proximal tubules leading to increased bicarbonate reabsorption from the distal tubule with a physiological inhibition of ammonia formation in the distal tubules. Milne, Stanbury and Thomson (1952) demonstrated the same mechanism in a case of the Fanconi syndrome. Both the cases reported in this paper had a low ammonia excretion. Most reported cases of this disease have been said to have an increased ammonia excretion. Stowers and Dent (1947) stated that their patient had a raised excretion on the basis of finding a raised ammonia index in the urine. This finding is now suspect because later patients whose urine was collected by more careful technique have not shown high ammonia indices on twenty-four-hour excretion (Dent, personal communication). Dent has also pointed out that the high amino acid content of these urines, and their constant neutrality or alkalinity, all favour bacterial contamination and artefact ammonia production from urea. Whatever the mechanism of the acidosis, the effect must be to drain base from the body, and in chronic acidosis the main cation to be excreted with the excess acid anion is calcium. Thus not only is there difficulty in precipitating

calcium phosphate complex into bone because of the low serum phosphate, but there is in addition an actual drain of basic calcium cations from the bones to neutralise the acid anions which the kidney has failed to deal with in the normal way.

The balance charts demonstrate the effect of large doses of calciferol given while the patients are in acidosis. There is a lowering of faecal calcium but there is an immediate and equal increase in urine calcium so that the balance remains negative. Calciferol therapy alone may raise the ionic product of calcium and phosphorus and permit some deposition of calcium salts in osteoid, as in Stowers and Dent's case; indeed considerable healing of the rickets in Case 1 had occurred during the past six years while he had had this treatment. Nevertheless the balance was negative; so one is not surprised that growth had been at the rate of one inch

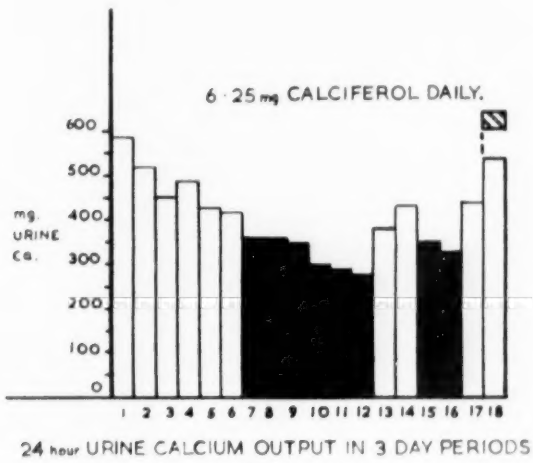

FIG. 4

Case 1—Histogram showing urinary calcium excretion in eighteen three-day periods. There was a gradually decreasing urinary calcium in the first six periods due to a wearing-off effect of calciferol which had been given up to six weeks previously. Sodium bicarbonate (black columns) caused a further decrease in urinary calcium. Discontinuance of alkalies in periods 13 and 14 caused an immediate increase in urinary calcium which was again reduced by alkalies in periods 15 and 16. When alkalies were stopped once more a further increase in urinary calcium occurred, and this appeared to be accentuated in period 18 by calciferol.

a year for the previous three years, and that the patient was a dwarf. It will be noted that this patient had a severe hypercaluria, and this is ascribed to the prolonged and massive doses of calciferol he had had in the presence of acidosis. It is no doubt this tremendous calcium loss in the urine that has practically corrected the acidosis. Despite the apparent mildness of the acidosis this patient needed 20 grammes of sodium bicarbonate daily to bring the alkali reserve within normal limits.

At this point it is worth considering the histograms of urine calcium excretion. In Case 1 (Fig. 4) they clearly show the gradual decrease in the very high calcium excretion before treatment—a wearing-off effect of the calciferol, which had been discontinued six weeks before balance studies were started. The decrease in urine calcium was also well seen after the administration of sodium bicarbonate; this was confirmed by discontinuing and recommencing the alkali, which caused concomitant rise and fall in urine calcium. In the histogram in Case 2 (Fig. 5) the steady urine calcium excretion at 200 milligrams daily will

be noted before treatment and the steady decrease when sodium bicarbonate was given. When one considers that this patient has osteomalacia, the excretion of 200 milligrams of urine calcium a day must be considered to be very large. Again from the histograms it can be seen that calciferol given alone caused a dramatic rise in urine calcium excretion but this was stopped when sodium bicarbonate—in Case 2 sodium bicarbonate and potassium bicarbonate—was given concurrently.

The usual treatment of acidosis in the Fanconi syndrome has been to give a sodium citrate and citric acid mixture (Shohl's solution), with the object of administering base orally without making the gut alkaline, because alkalinity was believed to hinder intestinal absorption of calcium. But this is illogical, for the reaction of the small intestine is normally alkaline. Furthermore Farquharson, Salter, Tibbetts and Aub (1931) showed that large doses of alkalies have no effect on calcium metabolism in normal individuals. They also showed that the administration of acid had no effect on faecal calcium excretion, although it increased urine calcium. We found that the administration of tablets was more convenient for the

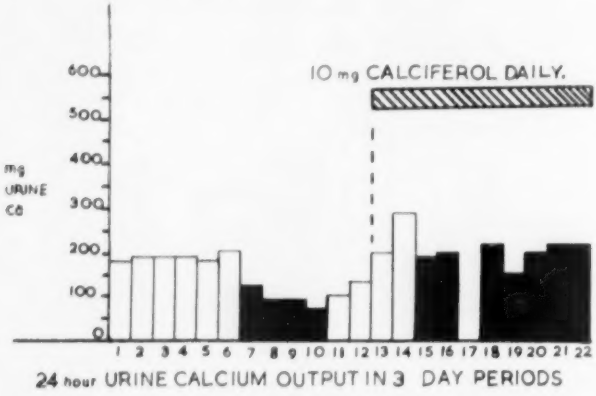
10 mg CALCIFEROL DAILY.
mg URINE Ca
600
500
400
300
200
100
0
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22
24 hour URINE CALCIUM OUTPUT IN 3 DAY PERIODS

FIG. 5

Case 2—Histogram showing urinary calcium excretion in twenty-two three-day periods. There was an excretion of 200 milligrams a day in the first six periods when the patient had no treatment. Sodium bicarbonate (black columns) reduced urinary calcium in periods 7 to 10 but when it was discontinued in periods 11 to 14 the calcuria gradually increased to previous levels, and in period 14 calciferol had caused a further increase to nearly 300 milligrams a day. Further alkali therapy in periods 15 to 22 again decreased urinary calcium output despite continued calciferol administration.

patients, and more accurate in dosage than a solution. We therefore gave our patients one gramme tablets of sodium bicarbonate or sodium and potassium bicarbonate. The balance charts show that these tablets had no adverse effect on intestinal absorption of calcium and phosphorus.

Large doses of sodium bicarbonate would be expected to lower serum potassium in a potassium-deficient subject. Berliner, Kennedy and Orloff (1951) showed that hydrogen ions compete with potassium ions in the tubule cells for exchange with sodium ions in the lumen of the tubules. When there are many hydrogen ions in the cells—as in acidosis—potassium ions tend not to be excreted in the urine. When the hydrogen ion concentration is lowered (pH rises) as by administration of sodium bicarbonate, more potassium is exchanged for sodium in the tubular lumen and so more potassium is excreted from the body.

Thus it can be seen that, in a person already deficient in potassium, the sudden correction of an acidosis, which itself tends to prevent the loss of potassium ions in the urine, is liable to cause further lowering of serum potassium by virtue of increased excretion in the urine

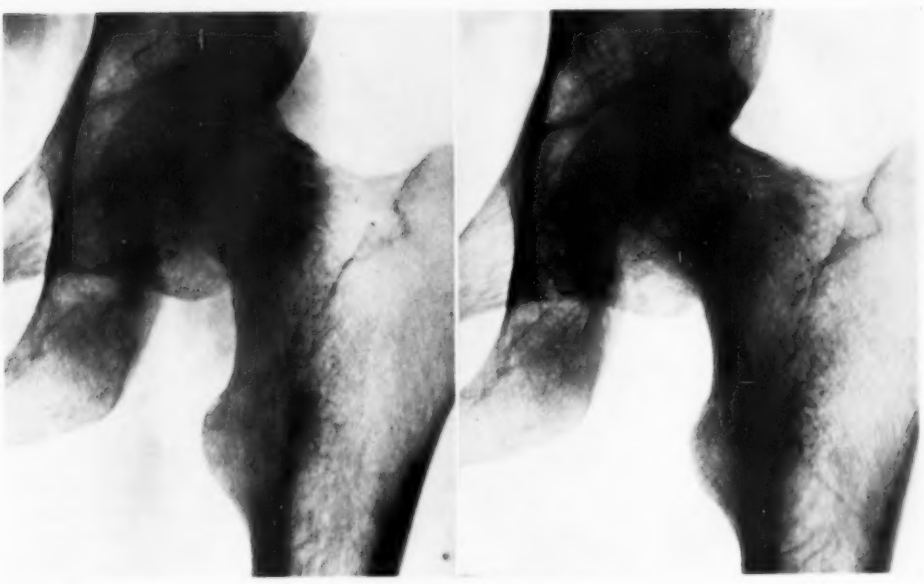

FIG. 6

Case 2—Radiographs showing fracture of neck of femur before treatment and the same bone six months later, after treatment.

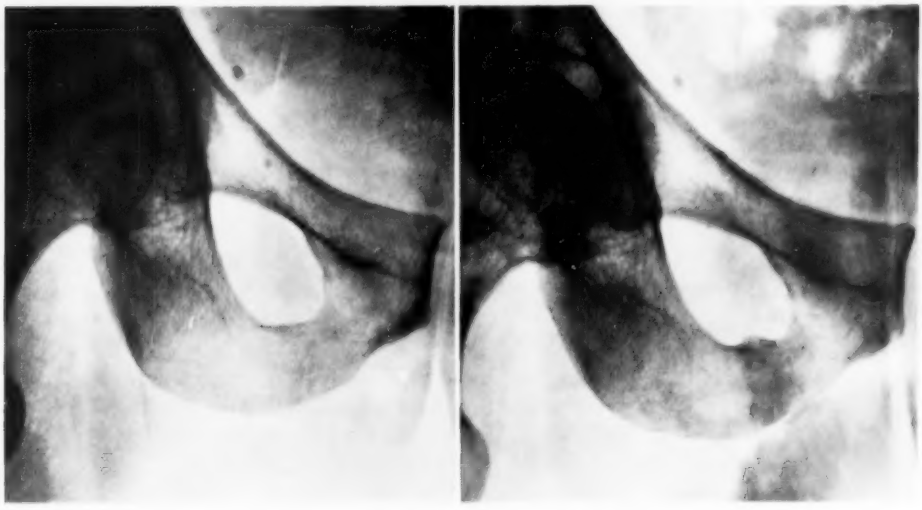

FIG. 7

Case 2—Radiographs showing pseudo-fracture of ischial ramus and the same bone after treatment.

as well as lowering of intracellular potassium by replacement of potassium in the cells by sodium. Both of our patients had lowered serum potassium and the man in Case 2 developed clinical symptoms with depression, abdominal distension, loose bowels, and marked weakness of the legs. These symptoms disappeared within a few hours of giving potassium by mouth, although his serum potassium had not then reached normal levels. We considered that this patient had a potassium-losing lesion of the tubules in addition to the other defects. We have continued to give him tablets containing potassium bicarbonate and sodium bicarbonate (1 : 4 w/w) as an out-patient for one year, with good results. When his acidosis had been corrected, as well as his hypokalaemia, his sense of well-being was remarkable. On looking back he realised his poor state of health before treatment. It is of interest that Debré's (1934) patient on alkali treatment died suddenly during a glucose tolerance test; and Guild's (1937) patient became distended and drowsy on alkalies so that his treatment had to be discontinued. Hypokalaemia was probably the cause of symptoms in all these cases and of death in one of them. Alkali therapy and large doses of glucose are both potent methods of lowering the serum potassium level.

When McCune, Mason and Clarke (1943) reviewed the literature they found more than half the patients were dead when reported. The prognosis was considered bad as regards life, and worse as regards health and ability to lead an active life. Of our patients the boy in Case 1 grew one and a half inches in six months after combined alkali and calciferol treatment and now leads a normal active life. The rickets is healed as judged from radiographs. The man in Case 2 took a job as a coal heaver after his discharge from hospital, " to test my bones out," as he put it. He has continued in his old job of stoker for the past eighteen months and works a fifty-six-hour week. The radiographs in this case (Figs. 6 and 7) show a rapid calcification of callus in the pseudo-fractures of the neck of the femur and of the ischial ramus. Calcification was present after six weeks' treatment and the patient was free from symptoms. It will be noted how easy it is to miss the ribbon-like osteoid zone in the pubic ramus before treatment and how obvious this becomes when calcified.

SUMMARY

1. Metabolic balance studies in two cases of the Fanconi syndrome are presented.

2. The actions of sodium bicarbonate and calciferol on the calcium and phosphorus balance were observed separately in the two cases.

3. The results show that sodium bicarbonate alone corrects acidosis and decreases the loss of calcium in the urine.

4. Calciferol in high dosage will increase intestinal absorption of calcium and phosphorus, but the urine calcium excretion then increases and vitamin D alone does not, therefore, give a positive balance adequate for complete healing and normal growth.

5. Alkalies and calciferol together put these cases into strongly positive calcium and phosphorus balance and promote healing of rickets, osteomalacia and pseudo-fractures.

6. Large doses of sodium bicarbonate in tablet form correct acidosis, do not adversely affect intestinal absorption of calcium, and facilitate accurate dosage and convenient administration.

7. Alkali therapy may lower serum potassium and precipitate symptoms of hypokalaemia in potassium-losing patients. This is thought to have been the cause of symptoms in several cases reported in the literature.

We wish to thank Dr C. E. Dent, Honorary Adviser in Metabolic Studies, for his advice and criticism during the investigation of these cases and for the original paper chromatograms on the urine in Case 1. We also wish to thank Dr T. Dixon and Mr J. W. Woollan for their help with the biochemical investigations. We record our thanks to Sister D. T. Fraser and the Nursing Staff of the metabolic unit, without whose care accurate balance studies would be impossible.

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THE DISTRIBUTION OF THE PERMANENT PARALYSIS IN THE LOWER LIMB IN POLIOMYELITIS

A Clinical and Pathological Study

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From the Institute of Orthopaedics and the Royal National Orthopaedic Hospital under a grant from the Medical Research Council

Though a striking feature of the paralysis that may result from an attack of poliomyelitis is its diversity, the belief that some order exists in the apparently irregular distribution of the permanent paralysis has been expressed by several authors. Wickman (1913) stated that " although a great variety of combinations of paralyses are found, certain types appear more often than others; in the leg the peroneal group and certain muscles of the thigh—in my experience the quadriceps femoris especially—tend to be implicated." Lovett and Lucas (1908), Lovett (1915, 1917), Jahss (1917), Mitchell (1925) and Legg (1929, 1937) showed tables indicating the relative frequency of paralysis and paresis in the muscles of the lower limb. All show a high incidence of paralysis in tibialis anterior, tibialis posterior, the long extensors of the toes and the peronei. A lower incidence of paralysis but a greater combined total of paralyses and pareses is shown in the quadriceps and in the gluteal muscles. No satisfactory explanation has yet been offered to account for these findings.

It is the object of this paper to review the distribution of paresis and paralysis in the muscles of the lower limb, to account for its disposition in terms of the destruction of motor nerve cells in the lumbo-sacral spinal cord, and to indicate the practical application of the findings in the management of poliomyelitis.

THE DISTRIBUTION OF PARESIS AND PARALYSIS IN THE MUSCLES OF THE LOWER LIMB

MATERIAL

The cases analysed in this paper were those in which a study of muscle recovery was previously reported in this Journal (Sharrard 1955). In 142 patients there were 203 lower limbs in which, three years after the onset of poliomyelitis, residual paresis or paralysis could be detected in one or more muscles by clinical examination. The seventeen muscles or muscle groups that are analysed here are the same as those described in the previous study of muscle recovery.

AFFECTION OF INDIVIDUAL MUSCLES

Of 2,464 affected muscles, 1,502 were paretic and 962 paralysed; the ratio of paresis to paralysis was 1·56 to 1·0.

The numbers of pareses and paralyses of individual muscles, and the proportions of paresis to paralysis in them, varied (Tables I and II). The main facts are that the quadriceps and the hip abductors (gluteus medius and minimus) lead in frequency of affection and in numbers of pareses, but the muscles of the leg are those most frequently paralysed. Tibialis anterior, tibialis posterior and the long flexors and extensors of the toes show low proportions of paresis to paralysis, while the hip flexors and hip adductors show a high proportion.

The order of the muscles shown in Table I confirms Lovett's (1915) finding that the muscles nearest the trunk are more frequently affected than the distal ones. He observes that " the demands on the hip and shoulder muscles are simple and less continuous than on the muscles of the lower leg and forearm or of the hand and foot. The latter are continuously

TABLE I

THE FREQUENCY OF AFFECTION OF 2,464 MUSCLES IN THE LOWER LIMB

Muscle	Number affected
Quadriceps	184
Hip abductors (gluteus medius and minimus)	178
Inner hamstring muscles	168
Biceps femoris	164
Hip adductors	162
Hip flexors	159
Tibialis anterior	152
Tensor fasciae latae	149
Gluteus maximus	142
Tibialis posterior	140
Flexor hallucis longus	133
Flexor digitorum longus	130
Extensor digitorum longus	130
Calf muscles (triceps surae)	129
Peronei	126
Extensor hallucis longus	125
Intrinsic foot muscles	93
Total	2,464

active in small fine complicated movements, whereas the large muscles nearest the trunk deal with coarser and less frequent movements." It is tempting, on this basis, to assume that a causal relationship exists between differences in the size and function of individual muscles and their frequency of affection by poliomyelitis.

When, however, the same muscles are arranged in order of frequency of *paralysis* (Table II) the picture is completely different. The largest number of paralyses are found in the distal muscles, which also agrees with Lovett's findings. To explain them he states that " the severity of the paralysis is proportionate to the weight to be met by the muscles at different levels, not because this factor influences in any way the original affection of the (nerve) cells but because it may retard the recovery of those muscles working against the greatest weight." Although this hypothesis may be attractive, it fails to explain several important discrepancies. There is a very low incidence of paralysis in the intrinsic foot muscles which, by Lovett's reasoning, should be the most frequently paralysed of all muscles in the lower limb. The number of paralysed inner hamstring muscles differs from the number

of paralysed biceps femoris muscles, though it should be about the same. Further, the rate of recovery in proximal and distal muscles, instead of showing the considerable differences needed to account for the complete dissimilarity between the order of the muscles in Tables I and II has been shown to be the same for all muscles in the lower limb (Sharrard 1955).

TABLE II

THE INCIDENCE OF PARALYSIS AND PARESIS IN THE MUSCLES OF THE LOWER LIMB. THE MUSCLES ARE SHOWN IN ORDER OF FREQUENCY OF PARALYSIS.

Muscle	Number paralysed	Number paretic	Ratio of paresis to paralysis
Tibialis anterior	103	49	0·47
Tibialis posterior	89	51	0·57
Flexor digitorum longus	85	45	0·52
Flexor hallucis longus	84	49	0·58
Extensor hallucis longus	66	59	0·89
Extensor digitorum longus	63	67	1·06
Peronei	60	66	1·10
Calf muscles (triceps surae)	57	72	1·26
Biceps femoris	54	110	2·03
Hip abductors (gluteus medius and minimus)	47	131	2·78
Quadriceps	46	138	3·00
Inner hamstring muscles	43	125	2·90
Tensor fasciae latae	38	111	2·92
Hip adductors	36	126	3·50
Hip flexors	34	125	3·67
Gluteus maximus	33	109	3·30
Intrinsic foot muscles	24	69	2·87
Total	962	1,502	

As Skinhøj (1949) observed in a similar study, there is no quality of the muscle such as size, function, position in the limb or phylogenetic development that can satisfactorily explain the frequent affection of some muscles and the high proportion of paralysis in others.

ASSOCIATED PARALYSES

That certain muscles appear to be paralysed or weakened together has been noted by several authors (Courtney 1896, Wickman 1913, Lovett 1915); their observations were, unfortunately, incomplete and relied on clinical impressions rather than on numerical analyses.

The muscle charts of the 203 lower limbs in this series were reviewed to find out whether those impressions were correct, and to reveal associations of paralysis that might otherwise be overlooked. Coincident sparing or absence of paralysis in muscles, which is equally important and likely to escape clinical observation, was also noted. Each muscle or muscle

group was paired in turn with every other muscle. The number of times that both were paralysed in the same limb, that both were not paralysed, or that one was paralysed and the other not, was noted. The number of times that coincidence or contrariety of paralysis could occur purely by chance was also calculated. Differences between the expected and the observed findings were worked out and submitted to statistical analysis.

The assessment of statistical significance by the x^2 test gave a strongly positive result for *all* muscle pairs, the association factor for the most strongly associated pairs being more than two hundred times the minimum figure for statistical significance. A muscle pair was considered to be very strongly associated if the association factor was more than one hundred and fifty times the minimum.

TABLE III

ASSOCIATED PARALYSES IN THE LOWER LIMB

Muscles in heavy type are very strongly associated; those in normal type are strongly associated

Muscle	Associated muscles
Hip flexors (psoas)	**Quadriceps, adductors**
Adductors	**Quadriceps, inner hamstring muscles, hip flexors**
Quadriceps	**Hip flexors, adductors,** inner hamstring muscles
Inner hamstring muscles	Adductors, quadriceps, hip abductors
Hip abductors (gluteus medius and minimus)	**Gluteus maximus,** biceps femoris, tensor fasciae latae
Tensor fasciae latae	**Hip abductors, gluteus maximus**
Gluteus maximus	Hip abductors, biceps femoris, tensor fasciae latae
Biceps femoris	Gluteus maximus, hip abductors, calf muscles
Calf muscles (triceps surae)	Biceps femoris, flexor digitorum longus
Flexor hallucis longus	**Flexor digitorum longus, extensor hallucis longus**
Flexor digitorum longus	**Flexor hallucis longus, extensor hallucis longus**
Extensor hallucis longus	**Extensor digitorum longus, peronei,** flexor hallucis longus
Extensor digitorum longus	**Extensor hallucis longus, peronei**
Peronei	**Extensor digitorum longus, extensor hallucis longus**
Tibialis anterior	**Tibialis posterior**
Tibialis posterior	**Tibialis anterior,** extensor hallucis longus
Intrinsic foot muscles	**Calf muscles,** peronei

The results are given in Table III. Some associated paralyses—for instance between the long toe extensors and the peronei or between the hip abductors (gluteus medius and minimus) and the gluteus maximus—might be explained by the closely allied functions of the muscles, as had been suggested by Lovett (1915). Other associations, such as those between the calf muscles (triceps surae) and the biceps femoris (first described by Bennett in 1952) or between the quadriceps, the hip adductors and the hip flexors cannot be accounted for so easily.

The absence of a strong association between the inner hamstring muscles and the biceps femoris or between the long toe flexors and the intrinsic foot muscles provides further evidence against the theory that associated function leads to associated paralysis of muscles.

SEGMENTAL INNERVATION IN RELATION TO AFFECTION OR PARALYSIS OF MUSCLES

If the paralysis in a muscle is not related to any feature of the muscle itself, there remains the possibility that it may be related to its innervation, or, more precisely, to the site and extent of changes in the motor cells of the anterior horn of the spinal cord.

Evidence of such a relationship is given by an enquiry into the segmental incidence of muscle affection. For each spinal segment, the total number of affected muscles that receive

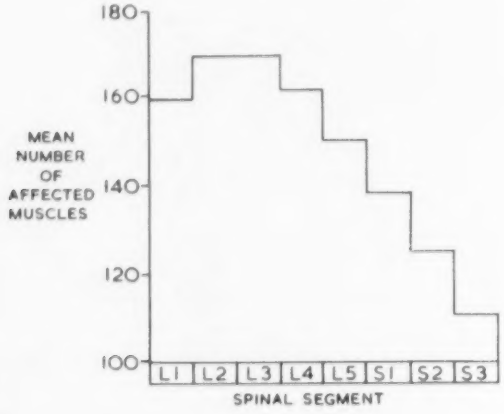

FIG. 1

The segmental incidence of affection of muscles in the lower limb.

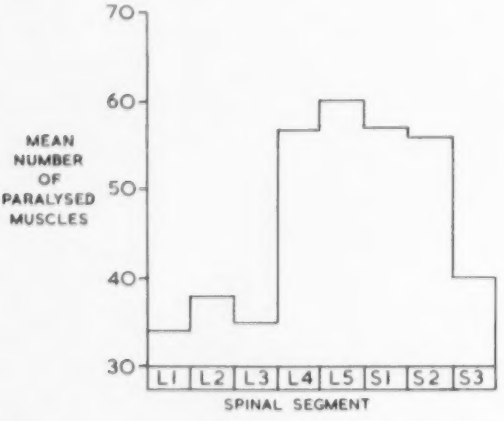

FIG. 2

The segmental incidence of paralysis of muscles in the lower limb.

a supply from the segment were noted; thus, in the third lumbar segment, the muscles were the hip flexors (159), the hip adductors (162) and quadriceps (184). The mean—in this segment 168—was calculated and used to plot a curve (Fig. 1), which thus represents the *segmental incidence of muscle affection*. The highest incidence is found in the second and third lumbar segments; below this level, there is a uniform decrease in the numbers of affected muscles that successive spinal segments supply.

The *segmental incidence of paralysis* (Fig. 2), derived in the same way from the data in Table II, is altogether different. It is included for comparison with an almost identical curve

obtained in the same way by Seddon *et al.* (1945) in an analysis of a large number of cases of poliomyelitis in the Malta epidemic of 1942–43. A high incidence is found in all spinal segments between the fourth lumbar and the second sacral segments; the curve, unlike that in Figure 1, is irregular and does not indicate that there is any direct relationship between segmental innervation and frequency of paralysis.

Attempts by earlier authors (Lovett 1915, Skinhøj 1949) to explain similar findings in terms of spinal cord lesions were hampered by the paucity of information concerning the localisation of function in the motor cells in man and by the incomplete accounts of the sites of destruction of these cells in poliomyelitis.

Five years ago a study of normal and poliomyelitic spinal cords was begun; the results, a summary of which will be given here, account for all the clinical phenomena of muscle affect, paralysis and association of paralysis described above.

THE SPINAL CORD IN THE NORMAL AND IN POLIOMYELITIS

METHOD OF STUDY

The principles underlying the study of the spinal cord in poliomyelitis are simple. Spinal cords were obtained from seven patients who had died at intervals varying between three months and eight years after the onset of the disease. The level of paralysis in the muscles of their lower limbs had been estimated by clinical examination and, whenever possible, confirmed at autopsy. The position and number of residual motor nerve cells in the lumbo-sacral spinal cord was determined and compared with the corresponding cells in the normal.

THE MOTOR CELL COLUMNS OF THE LUMBO-SACRAL SPINAL CORD

In practice, the analysis of spinal cords proved to be a formidable task. Previous descriptions of the topography of the motor nerve cells in the anterior horn of the grey matter proved, with few exceptions (van Gehuchten and de Neeff 1900, Bruce 1901, Romanes 1941, Elliott 1942), to be quite inadequate and a separate study of three normal spinal cords had to be made.

A method of reconstruction, by projection microscopy, of the nerve cell content of the lumbo-sacral spinal cord was devised (Sharrard 1953); serial sections were cut from the upper end of the twelfth thoracic spinal segment to the filum terminale, stained with eosin-azure, and the position, size and number of nerve cells in each successive series of twenty-five carefully superimposed sections plotted by drawing to produce a series of "cell charts." Each cell chart represents the nerve cell content of a 0·5 millimetre length of spinal cord. Examples taken from the third and fifth lumbar segments are shown in Figures 3 and 4.

The large motor nerve cells are arranged in columns whose disposition is remarkably constant at any given segmental level; the arrangement of the columns as seen in three dimensions is complex, and difficult to describe or illustrate. It would be inappropriate, in this article, to show the complete series of normal cell charts that were used as controls for the study of poliomyelitic cases; the detailed results that might be of value to other workers in this field will, it is hoped, be published later.

THE REPRESENTATION OF THE MUSCLES OF THE LOWER LIMB IN THE MOTOR CELL COLUMNS

Although previous work in experimental animals and in man suggested that the motor cell columns supply particular muscles or muscle groups, relatively little precise information was available about the representation of muscles in them in man. The sum of existing knowledge was well reviewed by Bok (1928). By comparing the presence or absence of cells in poliomyelitic spinal cords with the known activity in the muscles of the fourteen lower limbs concerned, much new information was obtained. The centres of innervation of all the

main muscle groups in the lower limb have been identified; in Figure 5 an attempt has been made to record the muscle representation in the cell columns at each segmental level.

In general, cell columns lying ventrally in the anterior horn supply the muscles in the proximal part of the lower limb, whereas those lying dorsally supply the more distal muscles

FIG. 3

Cell chart of the nerve cells in the grey matter of the third lumbar segment of a normal spinal cord.

FIG. 4

Cell chart of the nerve cells in the grey matter of the fifth lumbar segment of a normal spinal cord.

of the leg and foot. Flexor muscles are supplied by columns situated medial and caudal to those supplying the corresponding extensor muscles. Some muscles, such as the hip flexors, the hip adductors and quadriceps are supplied by long cell columns; others, such as tibialis anterior, tibialis posterior, flexor digitorum longus and flexor hallucis longus are supplied by

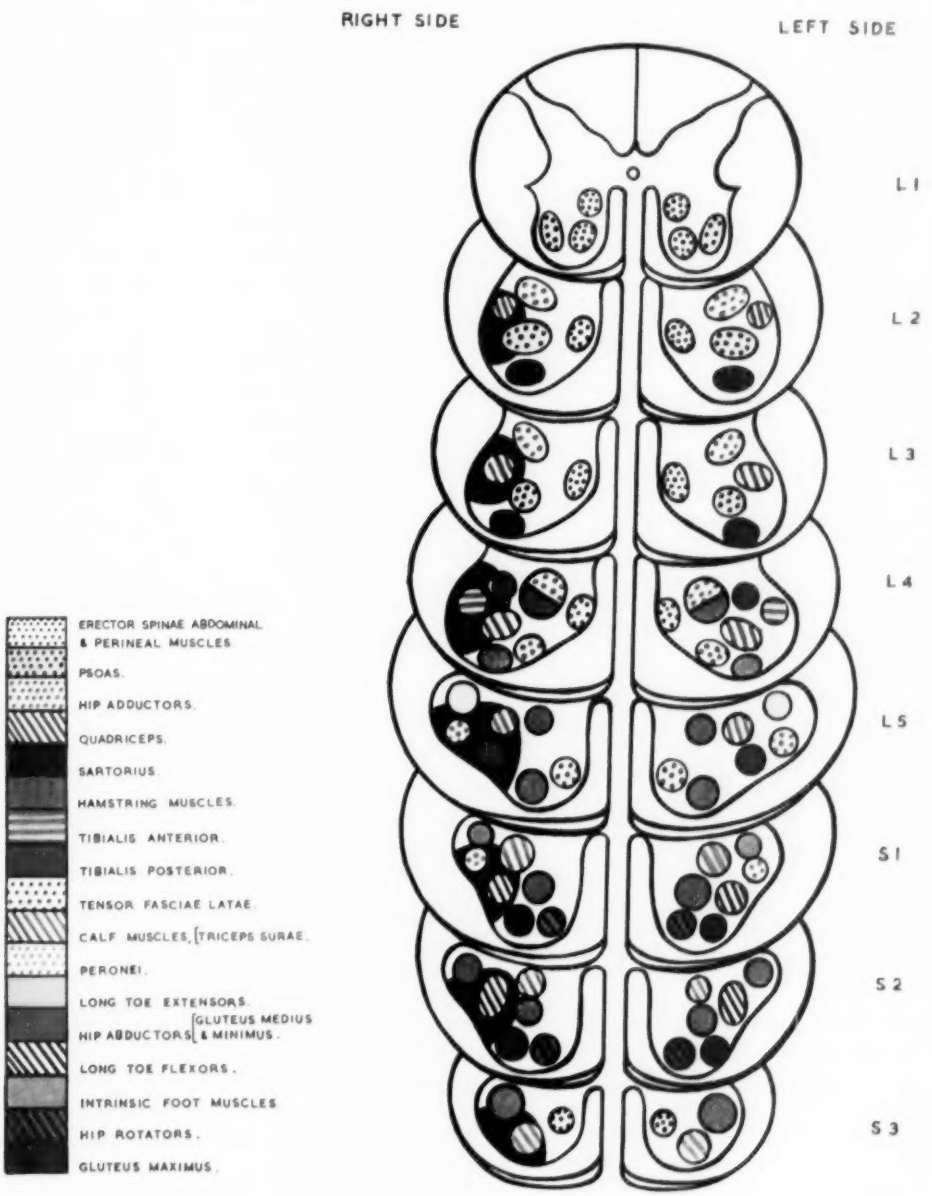

FIG. 5

The representation of the lower limb muscles in the motor cell columns of the lumbo-sacral spinal cord. The groupings of the columns are those that are the most characteristic in each spinal segment. Areas shown in black are those most liable to be affected by poliomyelitis.

short columns. It is important to note that, for two reasons, the root innervation of a muscle or muscle group does not bear an absolute relation to the length of its cell column. Firstly, a muscle may receive its supply from two spinal segments (for example, tibialis anterior—L.4, 5), but its cell column does not necessarily extend through the whole length of either segment. Secondly, lumbar segments are almost twice as long as their numerical counterparts in the sacral segments. Thus the cell column that supplies a muscle innervated by the first and second sacral segments is much shorter than one that supplies a muscle innervated by the first and second lumbar segments.

THE SITES OF MOTOR CELL DESTRUCTION IN POLIOMYELITIS

The general distribution of motor cell destruction in the anterior horns of poliomyelitic spinal cords, as distinct from the loss of cells in individual cell columns mentioned above, showed several constant features.

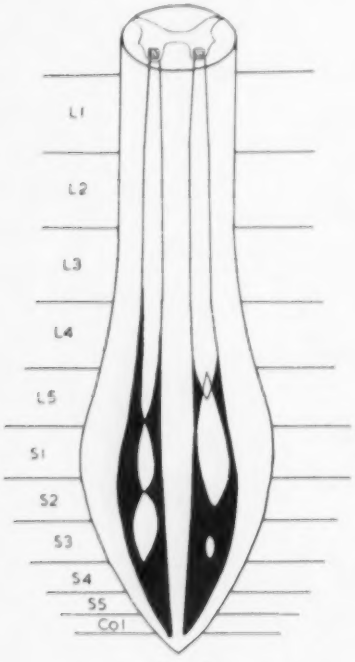

FIG. 6

A longitudinal analysis of the destruction of motor cells in the lumbo-sacral cord in a case of poliomyelitis with a moderate bilateral paralysis of the lower limbs. Regions shown in black contain residual motor cells. Note the complete absence of cells in the upper lumbar spinal segments and the focal loss in the lower lumbar and sacral segments.

TABLE IV

THE RELATIONSHIP BETWEEN MUSCLE POWER AND RESIDUAL MOTOR CELLS IN THE SPINAL CORD

Muscle power (M.R.C. scale)	Percentage of residual motor cells
0	0–2
1	2–3
2	3–5
3	5–10
4	10–20
4	20–40
5	over 40

The second and third lumbar spinal segments were the most frequently and extensively attacked in all the cords. Segments caudal to this were less often affected, the third and fourth sacral segments being especially likely to be spared. A similar segmental incidence of motor cell destruction has been noted by others (Horányi-Hechst 1935, Peers 1943, Elliott 1945, 1947).

Destruction was found not to be diffuse but localised in discrete foci of varying length and width with interposed lengths of grey matter of more normal cell content. An example of this is shown in Figure 6, which represents the longitudinal distribution of the lesions in one of the spinal cords, reconstructed from more than three hundred cell charts.

In the transverse plane the centre of the anterior horn appeared to be the most vulnerable area at most segmental levels. The fifth lumbar segment, in which the anterior horn has its greatest transverse area, demonstrated this central loss very clearly; the example shown in Figure 7 was typical of that found in many cell charts in the poliomyelitic cords at this level.

Motor cell destruction was always much more severe than would have been expected. One case in which there had never been any demonstrable weakness in any muscle in the lower limbs had suffered losses of up to 40 per cent of the normal number of cells in some cell columns. Far from there being any evidence that residual motor cells were functionally

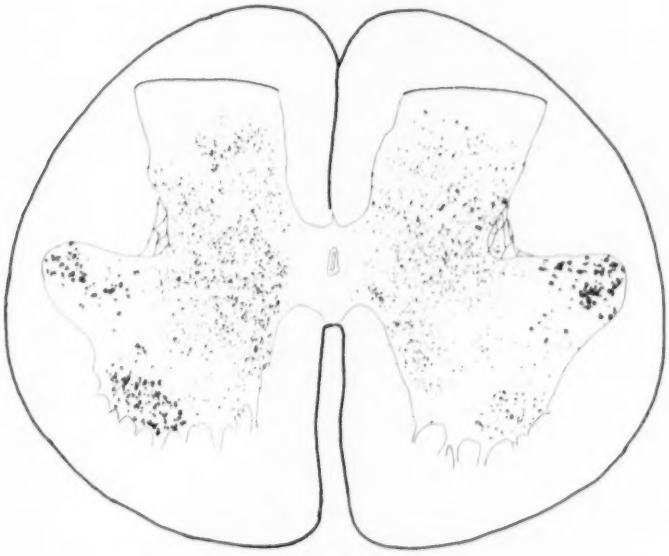

FIG. 7

Cell chart of the fifth lumbar segment of a poliomyelitic spinal cord. The centrally situated motor cell columns have been severely affected on both sides. (Compare with the normal in Fig. 4.)

inactive, it was surprising to discover how small a proportion of cells had been required to produce a useful contraction in the muscle they supplied. The residual power of a muscle was found to be closely related to the proportion of remaining motor cells that supplied it (Table IV).

THE RELATIONSHIP BETWEEN THE DISTRIBUTION OF THE PARALYSIS AND THE DESTRUCTION OF MOTOR NERVE CELLS

AFFECTION OF MUSCLES

The segmental incidence of muscle affection (Fig. 1) derived from the analysis of the clinical material agrees exactly with the general distribution of motor cell destruction described above. Since the upper lumbar spinal segments supply muscles in the region of the hip and thigh, while the lower lumbar and sacral segments generally supply the muscles of the leg and foot, it is easy to see why there is, apparently, a greater incidence of affection in proximal than in distal muscles in the limb. It is interesting to note that the hip muscle that derives its main supply from the sacral segments—that is, the gluteus maximus—is less frequently affected than other hip muscles (Table I). This is also true of the small external rotator muscles of the hip, though they are not shown in this Table.

The findings in the spinal cord also account for the large number of paralyses found in muscles such as tibialis anterior, tibialis posterior and the long flexor and extensor muscles of the toes. In Table V, the ratios of paresis to paralysis in individual muscles are compared with the lengths of the motor cell columns that supply them. Muscles supplied by short

columns of cells are the most frequently paralysed, those supplied by long columns are more likely to be paretic.

Figure 8 illustrates the probable mechanism responsible for this finding. A focus of motor cell destruction that severely affects the fourth and the upper part of the fifth lumbar spinal segment destroys almost all of the motor nerve cells that supply the tibialis anterior and most of those that supply the tibialis posterior. All other muscles are left with a proportion of residual motor cells sufficient to maintain normal power as judged by clinical examination.

TABLE V

THE RELATIONSHIP BETWEEN LENGTH OF CELL COLUMN AND THE RATIO OF PARESIS TO PARALYSIS IN MUSCLES IN THE LOWER LIMB

Muscle	Ratio of paresis to paralysis	Approximate length of motor cell column (*millimetres*)
Tibialis anterior	0·47	8
Flexor digitorum longus	0·52	8
Tibialis posterior	0·57	8
Flexor hallucis longus	0·58	8
Extensor hallucis longus	0·89	9
Extensor digitorum longus	1·06	10
Peronei	1·10	10
Calf muscles (triceps surae)	1·26	14
Biceps femoris	2·03	14
Hip abductors	2·78	16
Intrinsic foot muscles	2·87	15
Inner hamstring muscles	2·90	20
Tensor fasicae latae	2·92	16
Quadriceps	3·00	22
Gluteus maximus	3·30	17
Hip adductors	3·50	22
Hip flexors	3·67	23

The shape of the curve in Figure 2 can now be explained. The high incidence of paralysis in muscles supplied by the lower lumbar and upper sacral segments is due to the fact that most of them—tibialis anterior, tibialis posterior, peronei and the long muscles of the toes—are supplied by short columns.

All the features of the distribution of paresis and paralysis are satisfactorily explained in terms of lesions in the spinal cord. All the evidence is against a primary cause in the muscles themselves. If it were true that the distribution of paralysis was related to factors peculiar to certain muscles, movements or activities, evidence of it would be found in the disposition of cell destruction in the spinal cord. In fact, the focal loss of cells is completely independent of the boundaries of any one cell column; parts of cell columns are frequently obliterated in vertical or transverse planes. For the same reasons, the implication of higher centres as primary factors in the localisation of the paralysis is inadmissible.

There is, as yet, no adequate explanation for the greater incidence of motor cell destruction in the upper lumbar segments, and the progressively smaller incidence more caudally. In the transverse plane, the central sites of cell loss also found by Elliott (1945, 1947) in the anterior horn resemble those found by Krogh (1945) in experimental hyp-oxaemia of the spinal cord, and it is possible that vascular factors are partly responsible for them.

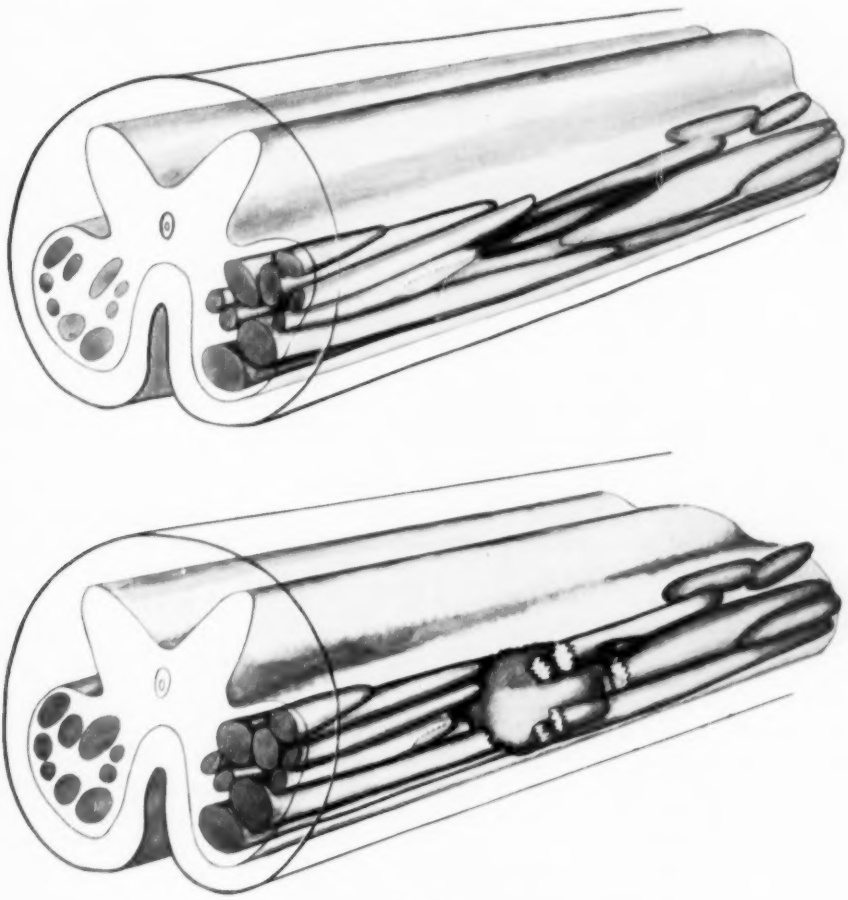

FIG. 8

A reconstruction of the cell columns of the third, fourth and fifth lumbar segments of the spinal cord. *Above*—the normal motor cell columns are shown; the two columns shown in colour are columns representing the tibialis anterior (blue) and tibialis posterior (yellow). The visible cut end of the cord is the caudal end. *Below*—all the motor cell columns in the lower part of the fourth and the upper part of the fifth lumbar segments have been obliterated by poliomyelitis. Only the short columns for the tibialis anterior (blue) and tibialis posterior (yellow) have been almost completely destroyed; only these two muscles will be paretic on clinical examination.

ASSOCIATED PARALYSES

When the whole of the motor column that supplied a muscle has been destroyed, it is likely that one or more adjacent motor columns that occupy the same length of spinal cord will be completely destroyed or severely affected. This is reflected in the clinical distribution of muscle paralysis. For instance, the columns that supply extensor hallucis longus, extensor

digitorum longus and peronei lie next to each other and occupy approximately the same length of spinal cord (Fig. 5). Paralysis of one of these muscles is, therefore, frequently associated with paralysis of the other two (Table III). Conversely absence of paralysis in one muscle is likely to be associated with absence of paralysis in the others.

Unusual associations, like that between the calf muscles (triceps surae) and the biceps femoris, can be accounted for in the same way, and would, indeed, be expected to occur, since their motor cell columns are so closely associated. The factor common to all strongly associated pairs of muscles is that their motor cell columns lie adjacent to one another and their segmental levels of supply correspond or overlap.

ASSOCIATED PARALYSIS AND THE PROGNOSIS FOR PARALYSED MUSCLES

In a previous paper (Sharrard 1955) the prognosis for a paralysed muscle was shown to be related to the degree of paralysis in muscles supplied by the same spinal segment. Associated paralysis between muscles may also be applied to determine prognosis. Ten muscles were chosen in which it may be important to know the prognosis for recovery. The probability of recovery to a functional power (M.R.C. grade 2, or greater) was worked out for each muscle 1) when both of its most strongly associated muscles were paralysed, 2) when one associated muscle was paretic or normal and the other paralysed, and 3) when both associated muscles were paretic or normal. The results are given in Table VI.

When both associated muscles were paralysed, the prognosis was very bad, particularly for the quadriceps, the hip abductors, tibialis anterior, tibialis posterior and the long toe extensors. When both associated muscles were paretic or normal there was an excellent prognosis for all muscles except tibialis anterior.

These results have lately been applied to the prognosis in individual patients and have been found to be valuable in making decisions about the management of the paralysis. A patient with paralysis of the quadriceps, hip adductors and hip flexors can be supplied, where it would be appropriate, with a caliper at an early date during the convalescent stage. The fear that the supply of an expensive instrument may be rendered invalid by recovery of the quadriceps will never be realised.

PATTERNS OF PARALYSIS IN THE LOWER LIMB

With the knowledge of the localisation of function in the motor cell columns that has been established above, it should be possible to deduce from the level of the paralysis in the muscles of any lower limb the approximate site and extent of lesions in the spinal cord, though not with such ease as in the diagnosis of a lesion in a peripheral nerve. Difficulties arise because, as Table IV indicates, a muscle is not paralysed unless the whole of its motor cell column has been affected. Even a residue of 10 per cent of intact motor cells may permit substantial activity in a muscle, and these intact cells may lie at the upper end, in the middle or at the lower end of the cell column.

There is another important difference between the analysis of peripheral nerve lesions and poliomyelitic lesions. Whereas lesions in peripheral nerves are much more often complete than partial in the transverse plane, lesions in the spinal cord frequently only involve part of the width of the grey matter at any given segmental level.

The distribution of muscle paralysis that might be expected to result from the existence of total or partial lesions of varying longitudinal extent in the grey matter of the lumbo-sacral spinal cord can nevertheless be worked out, and, conversely, all the common combinations or patterns of paralysis in poliomyelitic lower limbs can be explained in terms of one or more foci of motor cell destruction.

Figure 5 shows the position and size of cell columns for the lower limb muscles in the lumbo-sacral cord. The regions of the anterior horn that are shown in black in each segment are those most likely to be affected in partial lesions in the transverse plane. If all the cells

in the second, third and fourth lumbar segments are destroyed, only the quadriceps, hip adductors, sartorius and tibialis anterior will be paralysed; the psoas and tensor fasciae latae will be paretic, but other muscles will be normal (Fig. 9, right side). Should the destruction involve the fifth lumbar segment as well, the tibialis posterior will also be paralysed, the hip

TABLE VI

THE PERCENTAGE PROBABILITY OF RECOVERY IN MUSCLES IN RELATION TO AFFECTION OF THEIR ASSOCIATED MUSCLES

Muscle	Associated muscles	Both associated muscles paralysed	One associated muscle paretic or normal, the other associated muscle paralysed	Both associated muscles paretic or normal
Hip flexors	Quadriceps Adductors	21·6	68·1	98·7
Quadriceps	Hip flexors Adductors	Nil	10	78·9
Hip abductors	Gluteus maximus Tensor fasciae latae	Nil	15	89·4
Gluteus maximus	Hip abductors Biceps femoris	12·8	82·8	100
Tibialis anterior	Tibialis posterior Extensor hallucis longus	Nil	6·2	53·1
Tibialis posterior	Tibialis anterior Extensor hallucis longus	2	59·1	88·9
Extensor hallucis longus	Extensor digitorum longus Peronei	Nil	39·9	95
Extensor digitorum longus	Extensor hallucis longus Peronei	3·8	51·6	100
Triceps surae	Biceps femoris Flexor digitorum longus	6·6	73·4	93·8
Peronei	Extensor digitorum longus Extensor hallucis longus	11·4	78·6	93·4

abductors and semimembranosus will be severely paretic and the long toe extensors and peronei mildly paretic (Fig. 9, left side).

The common isolated paresis of the tibialis anterior results from a partial lesion in the lower part of the fourth and the upper part of the fifth lumbar segments (Fig. 8). When the

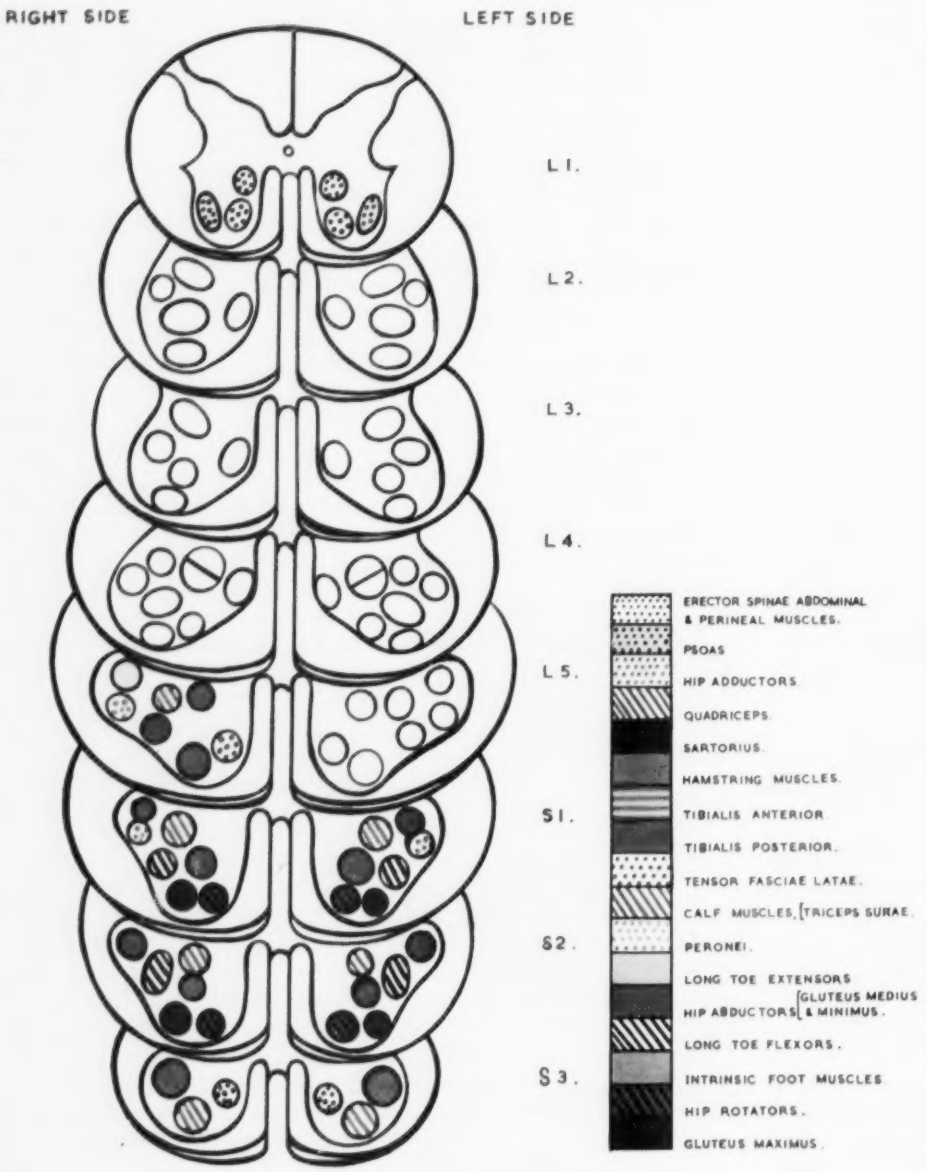

FIG. 9

A representation of motor cell loss in the lumbo-sacral spinal cord in poliomyelitis. *On the right side*, all the cells in the second, third and fourth lumbar segments have been destroyed. Cell columns for the quadriceps, hip adductors, sartorius and tibialis anterior have disappeared; these muscles will be paralysed. The psoas and tensor fasciae latae retain a portion of their cell columns and will be paretic. *On the left side*, the second, third, fourth and fifth lumbar segments have been destroyed. The cell columns for the quadriceps, hip adductors, sartorius, tibialis anterior and tibialis posterior have disappeared; these muscles will be paralysed. The hip abductors, semimembranosus, long toe extensors and peronei retain a portion of their cell columns and will be paretic.

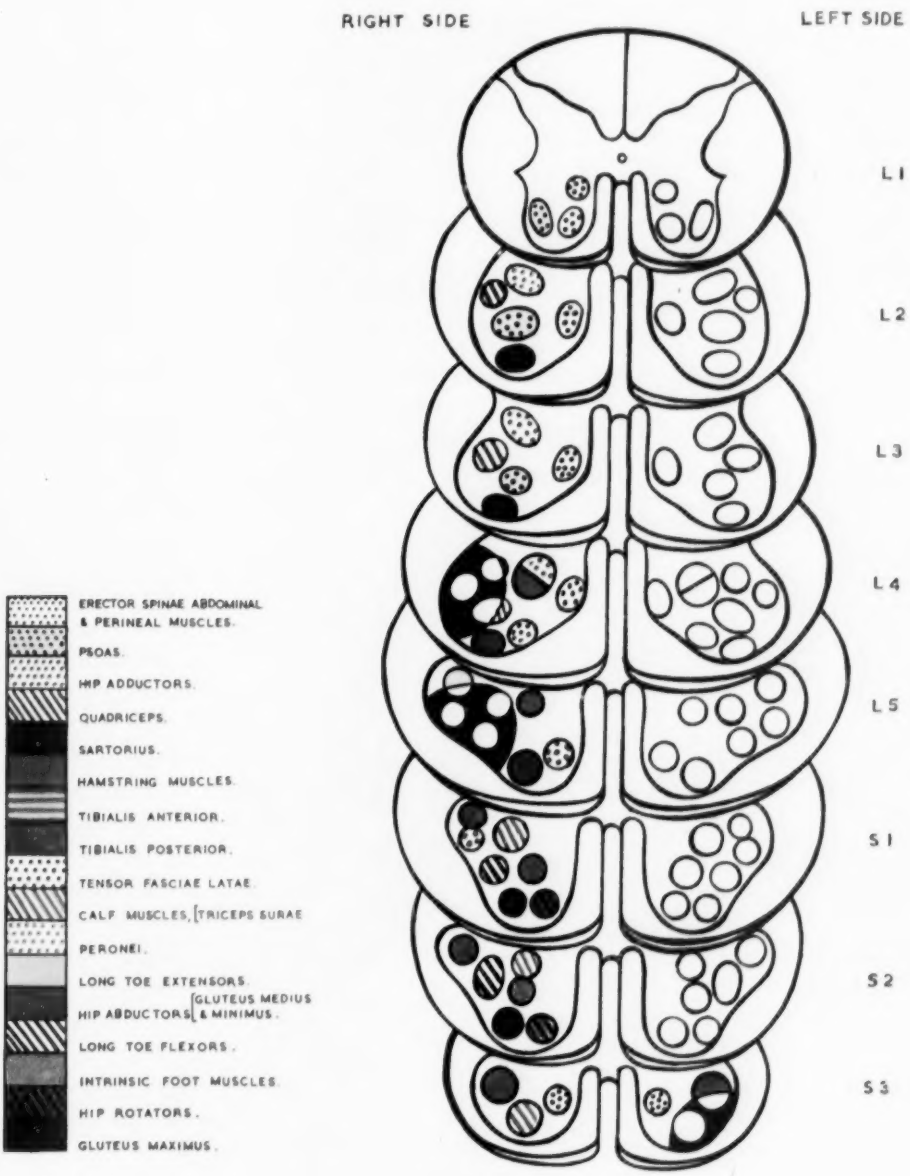

FIG. 10

A representation of motor cell loss in the lumbo-sacral spinal cord in poliomyelitis. *On the right side*, a partial lesion in the fourth and fifth lumbar segments has destroyed the cell columns for the tibialis anterior and tibialis posterior. All other muscles have retained a high proportion of their cells and the muscles concerned will not be clinically affected. *On the left side*, all the cell columns except those in part of the third sacral segment have been destroyed. Only a portion of the column for the intrinsic muscles of the foot remains. The clinical result will be a flail lower limb in which only the intrinsic muscles of the foot are active.

tibialis anterior and tibialis posterior are both paralysed, there is a partial lesion extending throughout both segments (Fig. 10, right side).

An extensive lesion that destroys all the motor cells in the lumbo-sacral segments gives rise to a flail lower limb in which only the intrinsic muscles of the foot and the peroneal muscles are active (Fig. 10, left side).

These few examples demonstrate how, by various combinations of destructive lesions in the grey matter of the spinal cord, the distinctive patterns of paralysis in poliomyelitis may be explained.

THE PRACTICAL APPLICATION OF THESE FINDINGS

Holdsworth and Hardy (1953) emphasised the importance of precise knowledge of the pathological lesion in the diagnosis, in the planning of treatment and in the assessment of the prognosis of traumatic paraplegia. In poliomyelitis the position is somewhat similar.

In the diagnosis of poliomyelitis at an interval after the acute stage, or when no febrile illness has been observed, the distribution of the paralysis alone may differentiate it from other lower motor neurone diseases. The paralysis in poliomyelitis is rarely precisely symmetrical. So frequently are the hip abductors, quadriceps or tibialis anterior affected that absence of paresis or paralysis in any of them in the presence of paralysis of other muscles in the lower limb makes a diagnosis of poliomyelitis unlikely. By contrast, in peroneal muscular atrophy the muscles most frequently affected in the early stages of the disease are the intrinsic muscles of the feet and peronei, a combination of paralysis rarely seen in poliomyelitis. Incidental to the comparison between the paralysis in these two diseases is the finding that the common bilateral symmetrical pes cavus, sometimes attributed to unrecognised paresis of the intrinsic muscles of the foot, is rarely the result of poliomyelitis. Even unilateral pes cavus, provided it is not a medial cavus (Pilcher 1955) or a calcaneo-cavus, is more often associated with spina bifida than with poliomyelitis.

The quantitative relationship between the loss of motor nerve cells and the residual power in muscles is particularly important in cases regarded as aparalytic in the acute stage of poliomyelitis. Although there may never have been any clinical paralysis, a considerable proportion of motor cells may have been damaged or destroyed. The nerve supply to some muscles, especially those supplied by short cell columns, may have been diminished by up to 60 per cent. Patients without paralysis are frequently allowed to walk within two or three weeks of the onset of the major illness. It is known that overstretching or over-fatigue can occur in muscles such as the hip abductors or tibialis anterior and may result in deterioration in power (Lovett 1915, 1917, Sharrard 1955). Paresis, previously undetectable, may be revealed later by the development of a limp or a valgus foot. It is probably wise, therefore, not to allow the resumption of full activity in an aparalytic case at too early a date, and to continue to look for evidence of paresis over a period of not less than six months.

In the management of the later stages—that is, after the first six weeks—the importance of accurate muscle testing has been stressed by Seddon (1955) and has been found to be of value in the prognosis of recovery in *paretic* muscles (Sharrard 1955). If the results of muscle testing are also used to reconstruct the probable site of major cell destruction in the spinal cord, the clinician will have a much clearer idea of what he is trying to treat, and what the chances are of recovery in *paralysed* muscles. The presence of associated paralyses (Table VI) may also help to define the prognosis for recovery of a paralysed muscle. Recovery can be expected in some muscles but the prolonged and uneconomic treatment of those that are beyond all hope can be avoided.

SUMMARY

1. The distribution of the permanent paresis and paralysis in the muscles of 203 lower limbs affected by poliomyelitis is analysed and related to the destruction of motor nerve cells in the grey matter of the lumbo-sacral cord.

2. The tibialis anterior and tibialis posterior and the long muscles of the toes are more often paralysed than paretic; these muscles are innervated by short motor cell columns. Muscles such as the hip flexors and hip adductors that are more often paretic than paralysed are innervated by long cell columns.

3. Muscles innervated by the upper lumbar spinal segments are more frequently affected than those innervated by the sarcal segments. This agrees with the segmental incidence of motor cell destruction found in poliomyelitic spinal cords.

4. Each muscle or muscle group is associated in paralysis with other specific muscles. For instance, the long toe extensors with the peronei and the calf muscles (triceps surae) with the biceps femoris. Associated muscles are innervated by adjacent motor cell columns. The probability of recovery in a paralysed muscle can be determined by reference to the degree of involvement in its associated muscles.

5. The distribution of the paralysis in an individual lower limb is determined by the site and size of foci of motor cell destruction. The cell loss in certain common patterns of paralysis is described.

6. The practical application of these findings is discussed.

I wish to acknowledge with thanks the help and guidance given to me by Mr H. J. Seddon, Director of Studies at the Institute of Orthopaedics, University of London, at which this work was undertaken under a grant from the Medical Research Council. I am grateful to Dr H. A. Sissons and the members of the department of morbid anatomy who have collaborated and assisted in the pathological aspects of this study. The detailed results of the study of the first three poliomyelitic spinal cords were included in a thesis submitted to the University of Sheffield for the degree of Doctor of Medicine (1954).

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THE RESULTS OF A REALIGNMENT OPERATION FOR RECURRENT DISLOCATION OF THE PATELLA

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Recurrent dislocation of the patella is a relatively uncommon condition, yet Marion and Barcat (1950) in a review of the world literature found almost a hundred different operations described for its treatment. In contrast to this profusion very few reports are to be found showing the long-term results of more than a few cases operated upon by any one procedure. The doubts which arise in these circumstances about the value of operations to retain the patella in normal articulation with the trochlear surface of the femur have been accentuated in recent years by an increasing number of surgeons who prefer to excise the patella for this condition (Swynghedauw and Laine 1945, McFarland 1948, Merle D'Aubigné 1950, Lacheretz 1951, Macnab 1952). Excision of the patella is based upon the belief that the patello-femoral joint is either so damaged by the recurrent dislocations or so congenitally deranged as to make severe and disabling osteoarthritis inevitable; removal of the patella is considered to be the best prophylactic against further dislocations and future osteoarthritis.

This paper reports the results of an operation to realign the extensor apparatus of the knee, with retention of the patella. The operation has been used at the Wingfield-Morris Orthopaedic Hospital for the last twenty years. The results suggest that it has much to commend it.

THE OPERATION

The rationale of the operation is based upon the finding that in many patients who suffer recurrent dislocations of the patella there is an increase in the angle, present to a slight degree in normal knees, between the line of the quadriceps muscle and that of the ligamentum patellae. This increased angulation is due to an abnormally lateral position of the tibial tubercle and results in a tendency to lateral displacement of the patella when the quadriceps is contracted. The realignment operation aims to correct this abnormality by placing the ligamentum patellae in direct line with the quadriceps. The patella is also brought lower because it is frequently found to be unusually high in these patients. A patella in such a position—the so-called patella alta—is denied the normal buttressing effect of the lateral femoral condyle which counteracts the tendency to lateral patellar displacement.

The first step in the operation is to mobilise the tibial tubercle with the ligamentum patellae attached. The lateral patellar retinaculum and the vastus lateralis are then freely incised and finally the tibial tubercle is reimplanted medial and distal to its original site. The retinacular incision, which does not penetrate the synovial membrane, is essential if the transplantation is to effect a realignment of the direction of pull of the quadriceps femoris and not merely to angulate the ligamentum patellae at its attachment to the lower pole of the patella.* In most of the operations reported here the deficiency in the antero-lateral part of the capsule was left untouched; in five cases it was filled with an aponeurotic transplant from the lax medial retinaculum. In one other case the lax medial retinaculum was reefed. After the wound has healed a groin-to-ankle plaster cylinder is applied with the knee straight and the patient is allowed to bear weight for six weeks, after which the plaster is removed and active knee flexion is begun.

* No originality is claimed for this operation; it corresponds closely to the procedure recorded by Hauser in 1938, yet the patient in Case 23 was operated upon by the late G. R. Girdlestone in 1933.

TABLE I

CLINICAL DETAILS OF TWENTY-THREE PATIENTS TREATED FOR RECURRENT DISLOCATION OF THE PATELLA BY TRANSPLANTATION OF THE TIBIAL TUBERCLE

Patient	Knee	Age at operation (*years*)	Dislocations before operation	Duration before operation (*years*)	Period of follow-up (*years*)	Functional enquiry			
						Pre-operative	Post-operative (final follow-up examination)		
						General functional limitations imposed by the affected knee prior to operation	Pain*	Feeling of security in the knee	Functional capacity
1	L.	11½	12	3	2	Constant insecurity	None	Complete. Feels stronger than the other knee	Full activities
2	L.	22	8	6	2	The only disability was the recurrent dislocations	Sensation of stiffness after prolonged sitting with knee in one position	Complete	Full activities
	R.		2	1			No pain	Complete	Full activities
3	L.	15	2	8	2	None, apart from the dislocations and quite frequent subluxations which caused momentary instability	Some aching in wet weather	Complete	Full activities
4	R.	17	7	2	2½	Reluctant to walk outside home because of constant feeling of insecurity. Limped badly, was reluctant to run	None	Occasionally feels a little insecure	Full activities
5	L.	14	3	1	2½	A feeling of weakness in the knee	None	Complete	Full activities
6	L.	21	20	7	2½	Constantly guarded his movements to prevent the patella dislocating. Avoided games. The knee was painful towards the end of the day	None	Complete	Full activities
7	L.	23	3	3	3	None, apart from the recurrent dislocations	None	Complete	Full activities
8	R.	32	5	5	3	The knee felt insecure and ached occasionally after prolonged standing. Avoided games	None	Complete	Full activities
9	R.	15½	2	2	3	The knee felt insecure when walking over rough ground	Occasional slight discomfort when weather changes or after prolonged sitting in one position	Rarely may get the feeling of impending subluxation experienced pre-operation	Full activities
10	R.	12	12	5	4	The knee felt weaker than its normal fellow	None	Complete	Full activities

11	L.	27	2	$\frac{6}{12}$	5	None, apart from the recurrent dislocations	None	Complete	Full activities
12	L.	26	30+	11	7½	A constant feeling of insecurity; was afraid to cross the road. Avoided all games	None	Complete	Full activities
13	L.	16	30+	2	7½	Felt apprehensive of an impending dislocation when kneeling. Avoided games	None	Complete	Full activities
14	R.	10	30+	7	7½	None, apart from the recurrent dislocations	None	Complete	Full activities
15	R.	12½	7	4½	8	None, apart from the recurrent dislocations	None	Complete	Full activities
16	R.	31	30+	9	9½	Constant feeling of weakness in the affected knee	Pricking sensation in the knee when the weather changes	Complete	Full activities
17	L. R.	13	10	4	10	A constant feeling of insecurity	Occasional slight discomfort in the knees in damp weather. These symptoms are decreasing	Complete except for a little difficulty in balancing when walking downhill	Full activities
18	L.	34	30+	16	13½	Did not feel safe to walk without a supporting crepe bandage. Constant feeling of weakness	None	Complete	Full activities
	R.		1	$\frac{3}{12}$		None, apart from the dislocation	None	Complete	Full activities
19	L.	18	6	3	13½	None, apart from the recurrent dislocations	Occasional aching	Complete	Full activities
20	L.	29	30+	20	14	None, apart from the recurrent dislocations	None	Complete	Full activities
	R.						None	Occasionally feels slightly insecure	Full activities
21	L.	20	5	6	14½	Avoided all games. Apprehensive about walking downstairs	Occasional discomfort before wet weather. No pain after exercise	The affected knee feels weaker than its normal fellow	Plays games, but is still apprehensive about walking downstairs
22	L.	20	10	2	19	Painful on standing. Walked with a stiff knee gait to prevent dislocation	Long standing causes retropatellar pain	"Nearly as strong as the normal knee"	Full activity except avoids jumping
23	L.	27	30+	20+	20	Unable to run, kneel or play games	In the last three years walking more than three miles has provoked some aching	Complete	Full activities (with the limitations imposed by above-knee amputation of the other lower limb)

* "None" implies a response in the negative to the question, "Do you ever feel any pain at all in your knee, even at the end of a full day's work, after long standing or walking or when the weather changes?"

DETAILS OF PATIENTS AND RESULTS

This paper is based upon the results of operations performed by the surgeons of the Nuffield Orthopaedic Centre, Oxford, on twenty-six patients (thirty knees). The results of three operations must be described as failures and the three patients will be considered separately; only one has been examined, but all are included in order to give as true a picture as possible.

In Table I will be found details of the remaining twenty-three patients (twenty-seven knees), who have been personally interviewed and examined. The Table shows how long each patient has been kept under observation and his own assessment of the pre- and post-operative functional state of the affected knee. It will be seen that sixteen operations have been followed up for five years or more.

FIG. 1

The lower limbs are held supported by the heels. The right knee has been operated upon and shows a recurvatum of 20 degrees.

The following is a summary of the findings as determined at the final follow-up examination.

No dislocation of the patella has occurred since operation in any of the twenty-seven knees. Three patients on direct questioning said that on a few occasions they have felt that the patella (Cases 2R, 21, 22) might be going to " come out " but it has never actually done so. A small synovial effusion was present in three knees (Cases 2R, 20, 22) only.

All except three knees regained a full range of active movement; patient 5 lacked 5 degrees of flexion, patient 18 lacked 10 degrees of flexion in the right knee, and patient 23 flexed from 175 degrees to 80 degrees. Patient 9 required a manipulation under general anaesthesia six months after operation in order to regain full flexion.

There was complete stability of the collateral ligaments in all knees; slight cruciate laxity was noticed for the first time after the operation in seven (Cases 1, 13, 14, 15, 17, 18L, 21).

The quadriceps muscle was powerful and well developed in all cases. In thirteen of the nineteen unilateral operations the patella was found to have remained at the lower level at which it had been placed at operation.

In most patients retropatellar crepitus was greater in the knee operated upon than in its fellow. In several joints this crepitus was very fine, and in five cases it was absent (Cases 1, 4, 17L, 18R, 19).

Four out of the five patients who were operated upon at between ten and thirteen years of age were found to have developed a noticeable degree of genu recurvatum (Fig. 1). This did not happen in five others who were between the ages of fourteen and sixteen years at the time of operation. In the five knees which showed the deformity (patients 1, 10, 14, 17) there was some laxity of the anterior cruciate ligaments. A study of the radiographs indicates that the recurved position results from a combination of a growth deformity of the upper end of the tibia and laxity of the soft tissues of the knee. Patient 1 was singular because the interference with normal growth added a valgus element to the recurvatum deformity (Figs. 2 to 5).

FIG. 2 FIG. 3

Case 1—Lateral radiographs taken with the knees in the position of greatest active extension. Figure 3 depicts the knee operated upon.

Kneeling on a hard floor caused pronounced discomfort in four patients and an unpleasant pricking sensation in two. These symptoms appeared to arise from the original site of the tibial tubercle.

The late development of osteoarthritis—In this study, the presence or absence of an important degree of osteoarthritis in the knees subjected to operation has been assessed principally from the physical findings and the symptoms. This is because, in deciding whether osteoarthritis will spoil the result of operation, it is pain and stiffness rather than radiographic lesions that are important to patient and surgeon alike.

Judged thus, the results have not been marred by the advent of clinically significant osteoarthritis so far. The three patients who have been followed the longest (Cases 21, 22, 23) are, however, suffering minor symptoms which almost certainly do arise in osteoarthritic lesions (Table I). It must be emphasised that none of these patients seeks any form of treatment for these symptoms or regards them as anything but slight; leading questions were required to elicit the presence of these discomforts.

FIG. 4

FIG. 5

Case 1—Antero-posterior radiographs. Figure 4 shows the unaffected knee. The new bone formation in Figure 5 marks the site of attachment of the transplanted tibial tubercle. Slight valgus deformity is present.

FIG. 6

Case 23—Condition of knee twenty years after transplantation of the tibial tubercle. Moderate osteoarthritic changes are evident.

A study of the radiographs of the knees operated upon reveals similar findings. Those illustrated in Figure 6 show the knee of the patient who has been followed for the longest period and represent the most osteoarthritic picture of the whole series.

The post-operative course of two patients gives some reason to believe that repositioning of the patella may in certain cases slow down or reverse the osteoarthritic process. In both these patients the knees operated upon are now much healthier than the joints not operated upon in which there is reason to suspect an unstable and untreated patello-femoral relationship.

CASE REPORTS

Case 15—This patient obtained an excellent result in her right knee, in which the patella had dislocated at least seven times before she came for treatment. On examination eight years after operation it was found that, with the knees flexed to 90 degrees or more, resisted extension produced considerable retropatellar pain in the left knee but not in the right, the one operated upon. On direct questioning it appeared that the left patella had never dislocated, but on two occasions may have subluxated; radiographs showed that, whereas the right patella looked normal, the left patella lay rather laterally, was of abnormal shape, and showed areas of subchondral rarefaction or cyst formation towards its lower pole.

Case 19—This patient was completely satisfied with the result of the operation on the left knee performed more than thirteen years before. There was no history of patello-femoral instability in the other knee, nor had it been injured, yet he considered that the knee operated upon was the stronger of the two. There is much more palpable crepitus in the unoperated right knee than in the left; a large osteophyte was felt arising from the edge of the trochlear surface of the right femur, and active and passive movements were diminished. When the right knee was flexed to 90 degrees the patella lay far laterally and seemed to be approaching a position of subluxation. On one occasion the patient was admitted to hospital for aspiration of a large synovial effusion from the right knee, and radiographs showed advanced narrowing of the patello-femoral space and extensive osteophyte formation involving femur, tibia and patella. The radiograph of the knee operated upon showed no abnormality except a patella of abnormal shape.

THE FAILURES

The following are the clinical details of the three cases classed as failures.

CASE REPORTS

Case 24—The fixation of the transposed tibial tubercle proved inadequate, and thirteen days after operation the wound was opened and the bone reattached. Two months later, however, the patella was found to be still displaced and it was excised.

Case 25—In 1947 the knee was operated upon at another hospital and by another technique. Dislocation recurred in 1947 and again five years later. The realignment operation was performed in 1952. On the twelfth day after operation it was found that the tibial tubercle had been pulled away from its site of implantation. The wound was therefore explored and the tubercle fixed by means of a bone peg engaging the posterior tibial cortex. The knee proved satisfactory for a year, but thereafter patello-femoral subluxation occurred on five occasions. The patella was later excised.

Case 26—A recurrently dislocating patella was but one of several congenital abnormalities in the lower limb. The realignment operation was successful for ten years but then the patella began to subluxate when the knee was extended, being well placed in the trochlear surface of the femur with the knee flexed.

DISCUSSION

The findings suggest that the realignment operation for recurrent dislocation of the patella gives a good chance of freeing the patient from both the disabling dislocations and from the sense of insecurity and that it will also allow good functional activity. The patient in Case 13 has written: "The result of the operation has been miraculous; it has removed me from constant dread and anxiety from which I had not been free for twenty-odd years. To be able to step out without fear is a blessing one takes for granted until one has been deprived of it. Now I can do it again!" The satisfaction expressed by this patient was repeated in a similar fashion by many of the patients under review.

Certain features common to the three failures in this series suggest that if at operation it proves difficult or impossible to lower the patella, extensor realignment alone is not likely to prove successful and medial transplantation of the tibial tuberosity should be combined with patellectomy.

Patello-femoral osteoarthritis has not been a clinical complication in the patients of this series. However satisfying this finding may be it must be offset by the observation that although sixteen patients have been followed for five years or more, the oldest is still under the age of fifty. Can the relatively healthy state of these joints be correlated with the observations made by McFarland (1948) and Macnab (1952) that recurrently dislocating patellae exhibit marked osteoarthritic lesions, such as fibrillated articular cartilage, denuded and sclerotic subchondral bone, and subchondral cysts? In a previous investigation of the pathogenesis of osteoarthritis (Harrison, Schajowicz and Trueta 1953) evidence was found to suggest that osteoarthritis develops when there is a markedly uneven distribution of pressure on articular cartilage for a long time. Such a state of affairs will certainly exist in these incongruous and displacing patello-femoral joints and will produce the lesions referred to above. If, however, abnormal mechanics can be set right, as by repositioning the patella, there are grounds for believing that the osteoarthritic changes may not only be halted but, by the obliteration of cysts and cartilaginous resurfacing, they may be effaced and repaired. Although patellar realignment probably can never restore joint anatomy to normal it is possible that in many patients the eventual condition of the joint will not cause symptoms sufficient to spoil the result of operation.

It is not possible to compare this particular operation with the many others that are in current use. With regard to excision of the patella, Brooke (1937) and others have written of the excellent function that can follow the operation when practised for fractures of the patella. Scott (1949), however, detected a considerable residual disability in patients in whom the patella had been removed for fracture when reviewed two to five years later. Knees that have sustained a fracture of the patella cannot be compared directly with those suffering recurrent dislocation of the patella. When the late results of patellectomy for recurrent dislocation become available it will be interesting to compare the functional capacity and the state of the joints with those reviewed here.

The development of genu recurvatum after operation in four out of five patients operated upon under the age of thirteen is disturbing. Marion and Barcat (1950) and Garnier (1950) both encountered this complication; Garnier's patient was fourteen years old at operation. In the five examples of recurvatum described here it seems likely that the tibial tubercle was transplanted far enough distally to disturb the muscle balance around the knee in favour of the quadriceps. In its turn this caused elongation of the posterior capsule of the knee and impaired growth of the anterior part of the upper tibial epiphysis; in one case the lateral part of the epiphysis was also retarded. In only one case was the tubercle moved in a progressively distal direction by growth; when this occurs it must presumably tend to increase the deformity. Macnab (1952) and Chandler (1952) referred to post-operative growth disturbances after transplantation of the tibial tubercle. It must be concluded that distal transposition of the insertion of the ligamentum patellae is better avoided under the age of fourteen years.

SUMMARY

1. A realignment operation is described for the treatment of recurrent dislocation of the patella.

2. In twenty-three patients (twenty-seven knees) operation prevented further dislocation. The physical and functional condition of the knees two to twenty years after operation is described.

3. Patello-femoral osteoarthritis was not a clinical complication in any of these patients. This finding is discussed.
4. Genu recurvatum developed in four patients operated upon between the ages of ten and thirteen years. The cause of this deformity is discussed. It is concluded that the realignment operation should not be performed under the age of fourteen years.
5. In three patients operation failed to control the recurrent dislocation.

I wish to express my thanks to Professor J. Trueta for suggesting this study to me, and to the surgeons of the Wingfield-Morris Orthopaedic Hospital, Oxford, and Mr G. Platt of the Royal Buckinghamshire Hospital, Aylesbury, for allowing me to examine the patients they have operated upon.

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DISPLACEMENT OSTEOTOMY OF THE UPPER END OF THE FEMUR

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The present position of surgery of the hip has encouraged me to review the results in a series of patients for whom I performed a displacement osteotomy during the years 1932 to 1952. This record illustrates the useful application of the procedure to three major conditions—osteoarthritis of the hip, old ununited fracture of the neck of the femur, and old unreduced congenital dislocation or subluxation of the hip.

It is fair to say that the operation is derived from that originally described by Lorenz (1925), of Vienna, for adult patients with unreduced congenital dislocation of the hip. He called it a "bifurcation osteotomy." The name was apt. The long lower fragment of the femur was realigned in the normal weight-bearing axis and the short upper fragment was made to lie at an inclination parallel to the slope of the pelvic wall; the angle where the two fragments met pointed into the acetabulum. As a matter of interest, fourteen of the forty cases recorded here were cases of old unreduced congenital dislocations or subluxations, and, although the technique of Lorenz was not followed exactly, Figure 1 is typical of the result ten years after operation.

During the 1920's patients with ununited fractures of the neck of the femur were treated in Liverpool by a similar osteotomy, but at the intertrochanteric level, with displacement inwards of the upper end of the shaft beneath the femoral head. Figure 2 illustrates a typical result fourteen years later.

McMurray (1939) described and used an oblique osteotomy with inward displacement of the femoral shaft for osteoarthritis of the hip. McFarland (1954) described the late results in a series of these patients. Twenty-one of my patients were operated upon for osteoarthritis; Figure 3 is a typical result eight years afterwards.

CLINICAL MATERIAL

Sixty-nine patients have been subjected to displacement osteotomy in the twenty years covered by this review. The average time since operation is eleven years. Forty of the sixty-nine patients have been examined during the last year; four are known to have died, and the remainder could not be traced. Of the forty patients that were examined twenty-one had osteoarthritis, fourteen congenital dislocation, and five ununited fracture of the neck of the femur.

RESULTS

The results have been assessed by the method described by Shepherd (1954) and the grading of the forty patients examined is shown in Table I.

TABLE I

Results of Displacement Osteotomy in Forty Cases

Excellent	Good	Fair	Poor
2	18	10	10

The results according to the patient's reply when asked whether the operation had been worth while are shown in Table II.

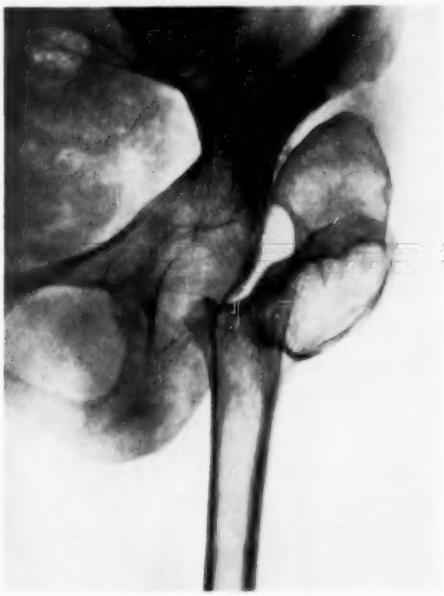

FIG. 1

Typical late appearance of realignment osteotomy for unreduced congenital dislocation of the hip. The patient was aged forty-five at the time of operation. Her pain has been relieved.

FIG. 2

FIG. 3

Figure 2—Late appearance of displacement osteotomy for ununited fracture of the neck of the femur. This patient has retained 90 degrees of flexion at the hip and walks without a stick. Figure 3—Typical late appearance after displacement osteotomy for osteoarthritis. The relief of symptoms in the left hip enabled the patient to return to work in spite of the condition of his right hip.

A high proportion of patients in all three conditions are therefore satisfied with their results over a long period. This suggests that the operation should continue to be given serious consideration and that the factors making for success ought to be established. No claim is made that the osteotomy restores movement to a hip (although incidentally this

TABLE II

PATIENTS' ASSESSMENT OF RESULT IN FORTY CASES

Enthusiastic "yes"	Qualified "yes"	"No"
31	4	5

may occur) but it is claimed that the previous range is retained, that pain is removed or relieved, that deformity is corrected, and stability ensured. Even in an assessment that includes an index of mobility half the patients had good results—a record not yet reached after a similar lapse of time by any method of arthroplasty.

DISCUSSION

Probably the common factor for success whether in a patient with osteoarthritis, ununited fracture of the neck of the femur, or old congenital dislocation of the hip, is that the upper fragment was adducted and has remained so. This change in position of the head-and-

FIG. 4

To show the reversal of the normal varus angle into a valgus angle.

neck fragment was noted by Osborne and Fahrni (1950); but their emphasis was on the alteration of the area of weight bearing of the femoral head and not upon adduction of the upper fragment which I believe so essential. This is the position in which nature heals a hip joint. In order to take advantage of it, and at the same time restore the femoral shaft to weight-bearing line, the normal varus angle between the neck and shaft must be reversed

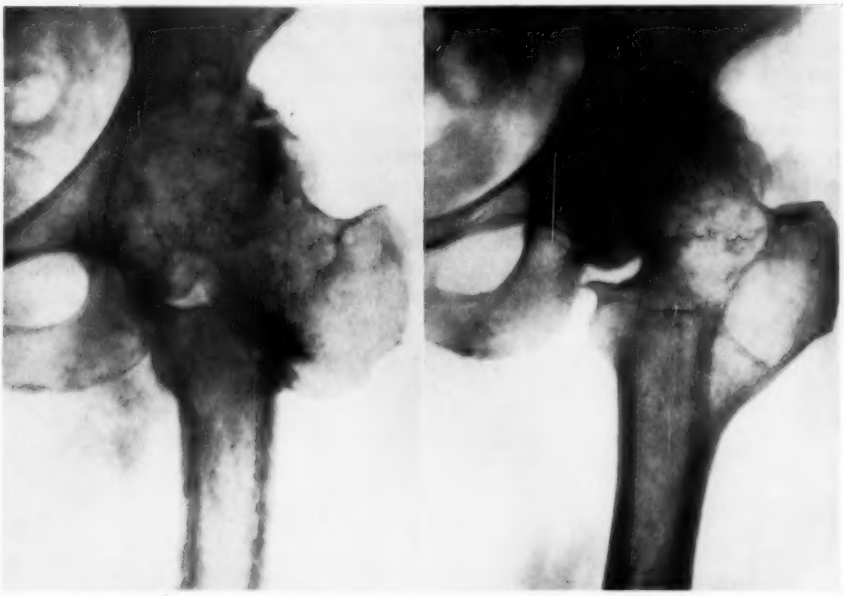

FIG. 5 FIG. 6

Case 1—Patient aged fifty-eight. Figure 5—Three months after displacement osteotomy for osteoarthritis. Figure 6—Eight years later. Note increase of cartilage space.

FIG. 7 FIG. 8

Case 2—Patient aged sixty-two. Figure 7—Before operation. Figure 8—Four years after displacement osteotomy. Note reappearance of cartilage space.

into valgus (Fig. 4). Failure to produce operatively this precise relationship, with the upper fragment adducted and the lower fragment in weight-bearing line, impairs the result. I have termed the establishment of this desirable position " reciprocal realignment." If the correct realignment is achieved one notices in late radiographs a reappearance of the joint space previously obliterated in the arthritic patients (Figs. 5 to 8). This tendency of the bone and articular cartilage of the femoral head to return towards normal after a successful osteotomy suggests a profound metabolic change. This was referred to by McFarland (1954). It is of interest also that in some cases of non-union of the femoral neck union of the fracture occurred after the osteotomy (Fig. 9).

Arguments adduced against the displacement osteotomy—The necessity to fix the hip in a plaster spica after operation is a disadvantage. No serious complications from this cause occurred in my series, but the patients were selected. The plaster spica prohibits the

FIG. 9

Case 3—Result six years after osteotomy for ununited fracture of the neck of the femur.

operation in just those elderly patients for whom it is most appropriate. Disabling stiffness of the knee is often quoted as another adverse feature, but this is not supported by the present series of forty patients. The average range of movement of the knee on the affected side was 100 degrees. Eight patients had a full range of movement and twenty-five had 90 degrees. The average age of the patients was forty-nine years at the time of operation.

Mention has also been made of valgus deformity of the knee on the side of operation. None of my patients has been affected in this way; and, although it was observed to a slight degree, it was not of any functional significance and it is not an inevitable accompaniment of the displacement. It results from abducting the leg more than is necessary. The statement that because of this genu valgum the operation should not be performed on both hips in the same patient cannot be supported; the deformity will occur only if the femoral shafts are over-abducted and immobilised for the whole period in that position. More importance should be attached to performing the osteotomy correctly. Experience leads me to believe

that most of the poor results are due to guessing the angle and site of the osteotomy and to the splintering that may occur at the inner cortex if it is divided blindly.

Technique of operation—Originally I used a straight incision beginning at the greater trochanter and extending five inches down the outer side of the thigh. The upper part of the shaft of the femur was exposed and divided just below the greater trochanter, the line of osteotomy running upwards and medially towards the lesser trochanter. With the osteotome still in position, the limb was abducted, the osteotome being used like a shoe-horn to displace inwards the upper end of the lower fragment. After closure of the wound a plaster spica was applied with the limb abducted to 45 degrees and the knee slightly flexed; the plaster included the foot. As a rule union was sufficiently advanced in two months for the patient to walk, though commonly a short plaster spica was applied for a further month or so.

I gradually modified this procedure in order deliberately to adduct the upper fragment, because, as I have indicated, a diseased hip left to natural processes tends to settle into

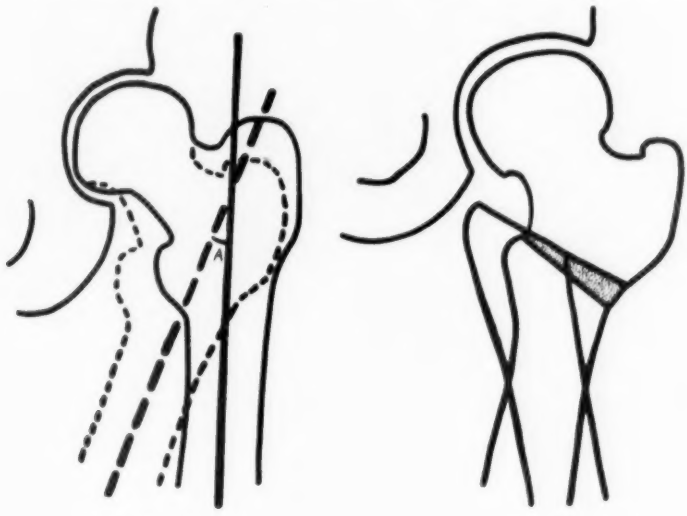

FIG. 10 FIG. 11

Figure 10—The angle A is measured before operation and the lower fragment is abducted through an equal angle at the site of osteotomy. Figure 11—If the hip has no free range of adduction the lower fragment is angled and displaced medially as shown.

adduction as it heals. Before operation the leg is adducted as much as possible and the angle that the shaft of the femur makes with the anatomical position is noted (A in Figure 10). This indicates the angle through which the shaft must be moved in the direction of abduction to take up the anatomical position against an upper fragment in full adduction. Since the upper fragment does not usually lie in the adducted position during the operation, after the osteotomy the shaft is abducted through angle A upon an upper fragment in neutral position. The hip is immobilised in this position. When union has occurred, and the leg is brought into anatomical or weight-bearing position, the upper fragment will be adducted and the reversal of varus into valgus attained (Fig. 4). If, before operation, the hip is already fixed in adduction it is only necessary to fix the limb, that is the lower fragment, in the neutral position after osteotomy.

If the hip has no free adduction movement or is fixed without deformity, then displacement and abduction must of themselves create the apex pointing to the acetabulum, and it may be necessary to remove some bone at the site of osteotomy to obtain this position

(Fig. 11). Figure 12 illustrates that when full adduction of the upper fragment is present or possible the line of osteotomy will be at right angles to the long axis of the femoral shaft. When no adduction is present the line of osteotomy becomes oblique (Fig. 13). These are extreme examples.

It is important to expose enough of the femur to be able to choose exactly the site of osteotomy. This varies with the lesion in question and should be so chosen that after displacement the apex of the new angle between neck and shaft will point at the lower border of the acetabulum. The site of osteotomy is therefore lower down the shaft in congenital dislocation, nearer the lower border of the greater trochanter in ununited fracture of the neck of the femur, and practically through the lower border of the trochanter in osteoarthritis.

FIG. 12

FIG. 13

Figure 12—When the upper fragment lies fully adducted the line of osteotomy is at right angles to the axis of the femoral shaft. Figure 13—When adduction of the upper fragment is absent the line of osteotomy is oblique.

The medial end of the line of osteotomy will be just below, through, or just above the lesser trochanter according to the obliquity required.

To ensure the desired adduction of the upper fragment, the correct degree of abduction of the lower fragment in relation to the upper fragment, and the precise selection of site and angle of osteotomy, the exposure was modified to attain a clear view of the anterior and lateral surfaces of the femur for one inch above and one inch below the intertrochanteric line, and to expose and make accessible the inner border of the femur above and below the lesser trochanter. The skin incision therefore extends from a point just below and lateral to the anterior superior spine downwards and backwards to the greater trochanter, and then turns down the middle of the outer side of the thigh for about six inches. The muscles attached to the anterior and lateral aspects of the trochanter and of the adjacent shaft of the femur are reflected from the bone sufficiently to give the exposure described above. The line of osteotomy is then marked as a groove across the front of the femur, and at its inner end a small osteotome is driven through the calcar femorale antero-posteriorly in the transverse plane. This

precludes the persistence of a spike of bone attaching to the lower fragment which can block displacement (Fig. 14). A broad osteotome is then driven from the lateral surface through the femur along the predetermined line. The leg is abducted and the osteotome is used as a lever to obtain displacement in the manner already described.

In order to avoid plaster immobilisation, and so make the operation suitable to those elderly patients for whom it is so clearly indicated, internal fixation has been increasingly

FIG. 14

Showing a spike of calcar femorale preventing adequate displacement of the shaft fragment.

used during the last three years. A nail-plate has been used which can be accommodated to the various angles required and yet is rigid enough to allow the patient to be nursed in bed without any external fixation.

SUMMARY

1. Forty patients out of sixty-nine operated upon between 1932 and 1952 by displacement osteotomy for congenital dislocation of the hip, ununited fracture of the neck of the femur, and osteoarthritis of the hip have been reviewed and the results analysed.

2. The factors responsible for the success of the operation are discussed and the technique of operation is described.

My thanks are offered to Professor Bryan McFarland for his helpful and constructive criticism, and to my registrars Messrs G. A. Wetherell and W. A. L. Thompson for interviewing and assessing my patients.

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FURTHER EXPERIENCES ON THE PATHOLOGICAL INFLUENCE OF A DEVELOPMENTAL NARROWNESS OF THE BONY LUMBAR VERTEBRAL CANAL

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In an earlier paper (Verbiest 1954) I reported some patients who developed signs of compression of caudal nerve roots on the basis of abnormal developmental narrowness of the bony lumbar vertebral canal. These signs appeared only during walking, standing or heavy bodily exertion. They disappeared immediately at rest. The symptomatology consisted of signs of irritation or of deficit. The most typical symptoms were one or more of the following: tiredness and loss of power in both legs, anaesthesia, a feeling of numbness in some lumbo-sacral dermatomes, or bilateral sciatica. The diagnosis had been made from: 1) myelographic examination; 2) the discovery at operation of a shallow canal and a compressed dural sac which did not contain cerebrospinal fluid and which expanded on decompression; and 3) the absence of other causes of compression.

In this paper further studies on the influence of a narrow lumbar spinal canal in the production of signs of compression of the caudal nerve roots will be described, with special reference, first, to the measurement of the width of the canal, and secondly, to the symptomatology of small disc protrusions when occurring in a narrow lumbar spinal canal.

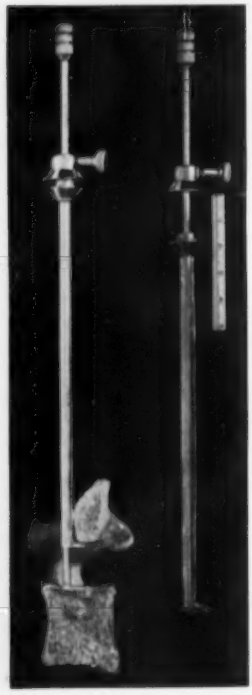

FIG. 1

Instrument for measuring the antero-posterior diameter of the vertebral canal.

A MEASURING DEVICE

In my previous paper I discussed the difficulties encountered in obtaining exact measurements of the spinal canal. The cross-section of the lumbar spinal canal has the form of a pentagon. Precise measurement would have to comprise the distance between the medial aspects of the pedicles of the vertebral arch, the length of these pedicles, the breadth of the laminae, the interlaminar angle and the mid-line antero-posterior diameter. From a practical point of view, however, there was no reason to measure more than the transverse interpedicular and the mid-line antero-posterior distances because these are at the moment the only ones that can be compared with values known for normal human skeletons (Huizinga *et al.* 1952). As they represent the length and breadth of the lumbar vertebral foramen, the measurements give some idea of the capacity of the lumbar vertebral canal.

In all my patients the interpedicular distance has been calculated from radiographs and, as previously reported, normal values were found. I could not obtain measurements of the antero-posterior diameter from the routine radiographs. Even sagittal tomographs were of little help because of scoliosis of the lumbar spine in most of the patients. It appeared, however, that this diameter could be estimated fairly exactly during operation. Since the ordinary calipers could not be applied for this purpose, a special instrument was designed by my assistants Brizzi and Kooyman (Fig. 1). For antero-posterior distance we measured the mid-line perpendicular from the arch to the body. Before measuring, the ligaments are carefully stripped from the vertebral arch. The posterior longitudinal ligament is not removed

because of the danger of profuse bleeding from the underlying veins. The inferior extremity of the measuring device is firmly pressed down against the posterior longitudinal ligament. The walls of the underlying veins are very thin and the thickness of the ligament opposite the body amounts as a rule to not more than three-quarters of a millimetre. The antero-posterior diameter is measured several times for greater accuracy. Although the measurements obtained by this method are somewhat less precise in the living than in the skeleton, the error is within one millimetre. The absolute range of variation in normals is 10 millimetres at L.5, 11 millimetres at L.4, 15 millimetres at L.3, 7 millimetres at L.2 and 8 millimetres at L.1. Narrowing is encountered most frequently in the area L.3–5. The measurements found with our method enable us to classify a canal as deep, shallow or possibly too shallow.

The following patient, in whom the measuring instrument was applied, is a typical case of developmental narrowing of the lumbar vertebral canal.

CASE REPORT

Case 1. *Narrowing of the spinal canal at L.5*—A man of fifty-three complained of sciatic pain in both legs when walking or standing for a long time. The pain disappeared rapidly at rest. Examination of the spine showed flattening in the lumbar region and some limitation of movement. The straight leg raising test was positive on both sides at 75 degrees. Plain radiographs showed tilting of L.4 and 5 to the left. *Lumbar puncture:* The fluid was clear and colourless; the Queckenstedt test was normal. Protein content was 60 milligrams per 100 cubic centimetres. *Myelography:* The fluid injected by cisternal puncture passed slowly to the level of the lower border of L.4, where there was an almost complete block. The lower border of the column had a toothed appearance. *Operation*—The spinal canal was narrow at the level of L.3–4 and very narrow at the level of L.5. After laminectomy in this area the dural sac was still compressed laterally by the articular processes between L.4–5. After removal of the medial part of these processes the dura expanded completely. Before laminectomy the antero-posterior diameters of the corresponding vertebral foramina were measured. These lengths were 14 millimetres at L.3, 12 at L.4 and 10 at L.5.* Comparing these values with those found by Huizinga *et al.* (1952) in normals, the antero-posterior diameters of L.3 and L.4 appear to belong to the small normal variants, while that of L.5 is definitely too small (Fig. 7).

THE EFFECT OF SMALL DISC PROTRUSIONS OR POSTERIOR SPONDYLITIC SPURS IN PATIENTS WITH A NARROW LUMBAR VERTEBRAL CANAL

CASE REPORTS

Case 2—A man aged forty complained of low back pain since falling from a roof in his twenties. Three months ago when riding on his bicycle he suddenly noticed a weakness in both legs. This sensation disappeared after a short rest. Since then he repeatedly complained of loss of power in the legs while walking or riding a bicycle. These disturbances always disappeared after a short rest.

There were no neurological symptoms. Examination of the spine showed a slight kyphosis in the upper, and an increased lordosis in the lower lumbar region. Plain radiographs of the lumbar spine showed a slight lateral shift of L.2 on L.3 and a lateral tilting of the upper lumbar vertebrae. The lateral view showed spur formation at the superior-posterior margin of the body of L.3 and of the inferior-anterior and posterior margins of the body of L.2. The second lumbar vertebral body was slightly wedged. *Lumbar puncture:* There was a slow response to the Queckenstedt test. The spinal fluid was clear and colourless. Protein was 30 milligrams per 100 cubic centimetres. *Myelography:* The fluid was arrested at the lower border of L.2. On the lateral view the contrast column was small at this point and the oil passed this level slowly by droplets (Figs. 2 and 3). The passage in the areas of L.2 and L.1 was also slow.

Operation—The antero-posterior diameter was 13 millimetres at L.1, 13 millimetres at L.2, 12 millimetres at L.3 and 16 millimetres at L.4. Opposite the spur at the superior border of L.3 and the inferior border at L.2 the diameter was only 9 millimetres, and at this level the dural sac was much compressed. After decompression the dural sac expanded. The post-operative course was uneventful.

Progress—The patient was completely relieved. Ten months after the operation he was free of complaints.

* All measurements of antero-posterior diameter presented in this paper have been determined by adding 0·75 millimetre for the thickness of the posterior longitudinal ligament to the values found with the instrument. Decimals have been rounded off upwards.

Comment—The antero-posterior diameters of L.2 and L.3 are equal to the smallest found in the normal, and that of L.1 is one millimetre smaller (Fig. 7). The intrusion of a spur of about three millimetres at the level of L.2–3 resulted in a narrowing of the vertebral canal to 9 millimetres. In this case the symptoms closely resembled those of developmental narrowing.

FIG. 2

FIG. 3

Case 2—Myelograph. Figure 2—Antero-posterior projection showing contrast medium arrested at the lower borders of L.1 and L.2. Figure 3—The lateral projection shows the constriction of the fluid at the level of L.2–3.

Case 3—A man of twenty-nine had complained of low back pain for nine years. During the past two years he had suffered from bilateral sciatica and weakness of both legs while standing or walking. These disturbances immediately disappeared in the recumbent position.

Apart from a bilateral positive straight-leg raising test at 80 degrees there were no neurological disturbances. Examination of the spine showed restriction of all lumbar movement. The low back pain was exaggerated by retroflexion. The spinous processes of L.5 and S.1 were painful on percussion. Plain radiographs revealed no abnormality. *Lumbar puncture:* The spinal fluid was clear and colourless. The Queckenstedt test showed a small and slow rise of pressure. Protein content was 33 milligrams per 100 cubic centimetres. *Myelography:* There was an arrest of the contrast medium at the level of the lower border of L.4. In the mid-line the fluid passed slowly by droplets to the termination of the dural sac.

Operation—The antero-posterior diameter was 15 millimetres at L.3, 11 millimetres at L.4 and 13 millimetres at L.5. The disc between L.4 and L.5 formed a small solid ridge across the spinal canal and protruded for not more than two millimetres. This resulted in a local narrowing of the lumbar canal to 9 millimetres, and at this level the dural sac was greatly compressed and did not contain fluid. Decompressive laminectomy of L.4–5 was performed.

Comment—The lumbar vertebral canal of this patient represented the smallest normal variation of L.4. At L.5 the antero-posterior diameter was only one millimetre longer than the smallest normal (Fig. 7). A small ridge formed by the intervertebral disc L.4–5 caused severe compression.

Progress—At the last examination four months after the operation the patient was free from complaints.

Case 4—A man of forty-two suffered for seven months from low lumbar pain radiating to the sciatic area of the right leg. This pain immediately disappeared in the recumbent position. Examination revealed a positive straight leg raising test on the right at 40 degrees. Straight leg raising on the left produced pain in the right leg. No other neurological symptoms were found.

The lumbar lordosis was obliterated. Movements of the lumbar spine were restricted in all directions. The spinous processes of L.4 and L.5 were tender on percussion. *Plain radiographs* showed lipping of the vertebral body of L.4, inclination of the lumbar spine to the left and rotation of the vertebral bodies to the left. *Lumbar puncture:* The spinal fluid was clear and colourless. There was a slow response to the Queckenstedt test. Total protein was 98 milligrams per 100 cubic centimetres. Myelography was not performed.

Operation—The spinal canal was narrow at the level of L.4–5. The antero-posterior diameter was 14 millimetres at L.3, 11 millimetres at L.4 and 13 millimetres at L.5 (Fig. 7). The disc between L.4–5 showed a small protruding solid ridge of not more than 2 millimetres. The distance between the lower border of the fourth lumbar arch and the ridge was 9 millimetres. The dural sac did not contain fluid at this level. Decompressive laminectomy of L.4–5 was performed and the medial parts of the encroaching articular processes were also removed.

Progress—At the last examination three months after the operation the patient was free from complaints.

Case 5—This patient, aged forty-seven, was a brother of the patient described in Case 1. He had complained of low lumbar pain for six years. This pain had its onset when he lifted a heavy weight. After a fall he suffered from pain in the left sciatic area, aggravated by sneezing and coughing, but present only while he was walking or standing and disappearing immediately at rest. *On examination* straight leg raising was impaired on the left. Straight leg raising on the right elicited pain in the left sciatic area. No other neurological signs were present. The lumbar lordosis was obliterated. All movements of the lumbar spine were restricted. Retroflexion accentuated the pain. *Plain radiographs* of the lumbar spine showed no abnormality. *Lumbar puncture:* The fluid was clear and colourless. There was a normal response to the Queckenstedt test. Total protein was 45 milligrams per 100 cubic centimetres.

Myelography—There was a block at the level of the lower border of L.4. After some hours part of the contrast medium had descended to the terminal part of the dural sac (Fig. 4).

FIG. 4

Case 5—Myelograph taken a few hours after the injection of the contrast medium. Arrest opposite the lower border of L.4. Part of the contrast medium has descended to the terminal part of the theca.

Operation—The antero-posterior diameter was 12 millimetres at L.4 and 14 millimetres at L.5. Thus the canal was narrow at these levels although not abnormally so (Fig. 7). The intervertebral disc formed a solid ridge which protruded about two or three millimetres into the spinal canal. Decompressive laminectomy of L.4 and L.5 was performed.

Progress—At the last examination four months after the operation the patient was free from complaints.

Case 6—A man aged fifty-four had suffered for nine years from paraesthesia and a sensation of numbness in both legs, occurring when standing or walking and disappearing immediately at rest. Six months before his admission to hospital he suddenly experienced, while working, severe low lumbar pain, radiating down the left leg in the sciatic area. On examination, the ankle jerk was absent on both sides. There was a bilateral slight paresis of the calf muscles. After myelography a complete paralysis of the left anterior tibial, extensor hallucis, and peroneal muscles developed. Both feet and the antero-lateral side of the left leg were hypoaesthetic. The lumbar curve was obliterated and lumbar spinal movement was limited. The spinous processes of L.4 and L.5 were tender on percussion. Plain radiographs showed lipping of the bodies of L.3, L.4 and L.5. *Lumbar puncture:* The spinal fluid was clear and colourless. The Queckenstedt test was positive. Protein content was 24 milligrams per 100 cubic centimetres.

Myelography—The contrast medium showed a block at the lower border of L.2. During the following hours part of the oil descended to the lower border of L.4, where there was a complete block. On the lateral view the contrast medium arrested opposite L.2 had a pointed inferior limitation.

The accumulated contrast medium opposite the body of L.4 gives an impression of the shallowness of the canal (Figs. 5 and 6).

Operation—The lumbar vertebral canal was very narrow at the level of L.2–5. Decompressive laminectomy was performed in this area. The medial parts of the encroaching articular processes were also removed. The disc between L.4–5 formed a small ridge protruding about three millimetres into the spinal canal. This small elevation contributed considerably to the compression of the dural sac as the canal was already narrow. The antero-posterior diameter was 13 millimetres at the superior and 11 millimetres at the inferior border of the arch of L.2, and 11 millimetres at L.3–5.

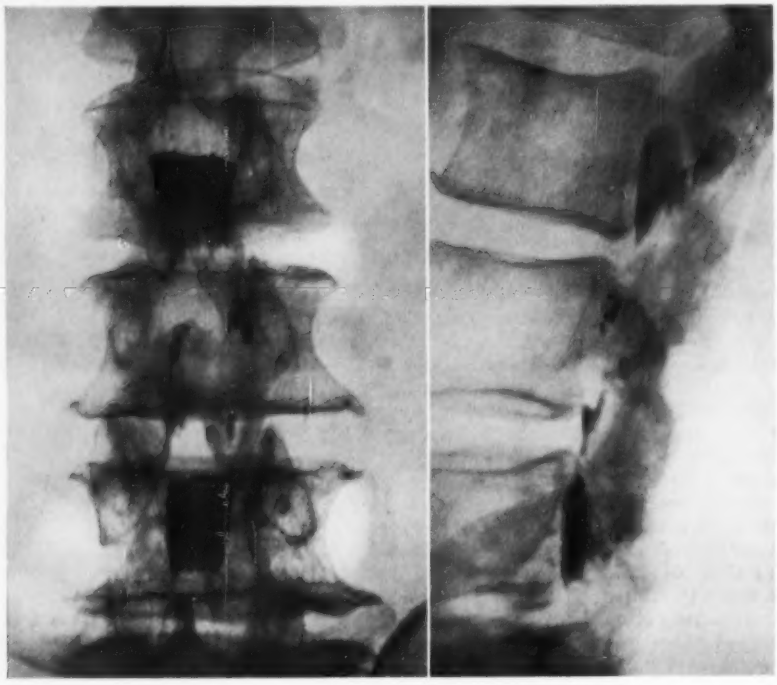

FIG. 5 FIG. 6

Case 6—Myelographs showing incomplete arrest of the contrast medium opposite the lower border of L.2 and a complete block opposite the lower border of L.4.

Comment—The antero-posterior diameters at L.2–5 were less than the smallest normal (Fig. 7). The history of eight and a half years of typical complaints of developmental narrowness of the lumbar canal are in accordance with these findings. A small disc protrusion of not more than two millimetres reducing the antero-posterior diameter to 9 millimetres produced signs of lumbo-sacral root compression simulating a tumour.

The interpedicular distance—Of the thirty interpedicular distances measured in these six patients, twenty-three corresponded to the mean or higher values found in normal skeletons; four varied from 1·4 to 2·5 millimetres below the mean and from 1 to 3 millimetres above the minimal normal values. Only the interpedicular distance at L.2 in Case 2 was abnormal, being 1·5 millimetres more than the corresponding maximal normal value (Table I).

DISCUSSION

The foregoing observations enable me to draw the following conclusions.

1) *Developmental narrowing of the lumbar canal caused by an abnormally short antero-posterior*

diameter does exist—In Case 1 the antero-posterior diameter of L.3–4 corresponded to normal lowest values, but at L.5 the diameter was definitely too small.

In Case 6 the antero-posterior diameter was abnormally small at L.1–5. At the level of L.4 the antero-posterior diameter corresponded to the lowest normal value. As there were no signs of bone disease both cases demonstrated a developmental narrowness of the lumbar spinal canal. These patients presented a typical symptomatology as described in my earlier paper.

2) *In the presence of a narrow, although not abnormally narrow, lumbar vertebral canal additional slight deformities, such as posterior lipping or small disc protrusions, can produce symptoms of compression*—The first suggestion for this conception was furnished by the experience with

TABLE I

INTERPEDICULAR MEASUREMENTS RECORDED IN NORMAL SKELETONS (HUIZINGA *et al.*) AND IN CASES 1–6. (Measurements in millimetres)

	L.1	L.2	L.3	L.4	L.5
Shortest	19	20	20	19	20
Longest	28	26	28	29	32
Mean	23·4	23·4	23·5	23·5	23·8
Interpedicular distance in Cases 1–6					
Case 1	26	24	23	24	30
Case 2	26	27·5	25	27	31
Case 3	22	22	22	25	32
Case 4	23	25·5	25	24	24
Case 5	25	23	21	27·5	27·5
Case 6	24	26	25	26	24

Case 7 reported in my previous paper. This was the only patient at that time in whom the antero-posterior diameter was measured. The values found were low, but not abnormally so. Taking the operative findings into consideration, I thought it justifiable, in that case, to ascribe the cause of the abnormal narrowness to encroachment of the heavily developed articular processes on the vertebral canal. Cases 2, 3, 4 and 5 presented in this paper support this conception. At some level the spinal canal of these patients presented the smallest antero-posterior diameter and probably such a narrowness alone would not have caused symptoms. A small protruding deformity at these levels, however, resulted in disturbances. In these patients the apparent contrast between the severity of complaints and the scarcity of neurological signs was most impressive.

In Cases 2 and 3 the clinical picture was similar to that of developmental narrowness. In Cases 4 and 5 the symptoms appeared in only one lower extremity, but in accordance with the symptomatology of developmental narrowness, both patients complained of sciatic pains only while standing or walking and these pains immediately disappeared at rest.

Three patients also complained of low lumbar pain (Cases 3, 4 and 5) and in Cases 3 and 5 low backache preceded the symptoms of radicular pains by seven and four years respectively. The low back pain was exaggerated by retroflexion in Cases 3 and 5. Movement of the lumbar spine was restricted in Cases 3, 4 and 5.

All the patients showed some deviation of the curvature of the lumbar spine. Neurological signs were few in all patients: positive straight leg raising test on both sides in Case 3 and on one side in Cases 2 and 4, and an increased total protein content of the spinal fluid in Cases 4 and 5. Myelography showed an almost complete block at the level of the protrusion.

The surgeon who does not take the narrowness of the canal into account may be astonished at the fact that the presence of such a small disc protrusion has been the cause of a complete block during myelography.

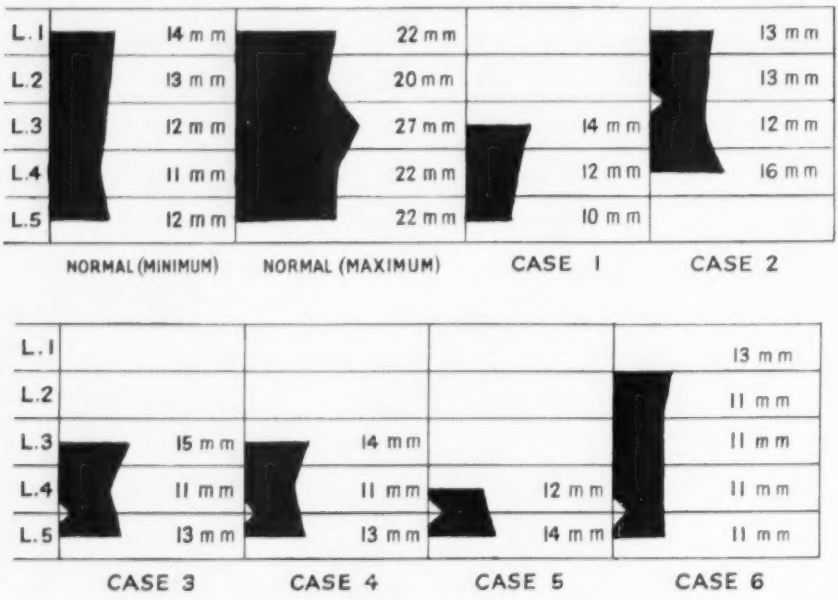

FIG. 7

Chart showing, in the first two columns, the minimal and maximal antero-posterior diameters of the lumbar vertebral canal in normal skeletons (Huizinga *et al.* 1952) and the antero-posterior diameters in Cases 1–6.

3) *In the presence of an abnormally narrow lumbar canal, a small disc protrusion may cause considerable damage to the caudal nerve roots*—This is demonstrated by Case 6. The antero-posterior diameters at L.2, 3 and 5 were definitely too small and that at L.4 was the smallest normal. For the last nine years this patient had suffered from typical disturbances as encountered in developmental narrowing of the lumbar canal. The presence of a small disc protrusion between L.4–5 had caused persistent disturbance of motor and sensory functions from damage to the lower lumbar and sacral caudal nerve roots.

The nature of the narrowing—From the data collected in this paper it appears that the narrowness depends mainly on shortness of the antero-posterior diameter. No sign of bone disease has been found. The fact that the patients in Cases 1 and 5 were brothers gives support for the hypothesis that the lesion is developmental. It may be that wedging or posterior lipping of the vertebral bodies, as found in our patients, bears some relation to developmental narrowness of the spinal canal because a similar association has, for instance, been described several times in achondroplasia (Spillane 1952). As in our previous series, the narrowness was most pronounced in the area where normally the canal is at its narrowest. As before, all patients were men. I want to stress the fact that the ligamentum flavum was carefully examined in all cases and that this ligament did not show any abnormal thickness

or hypertrophy. Consequently I do not think that in my patients the ligamentum flavum played a special role in provoking the disturbances.

Medico-legal aspect—The patients in Cases 2, 5, and 6 blamed a heavy exertion during work or an injury as the cause of their disability. Judicial interpretation will have to take into account that in such patients the narrowness of the canal is the main factor determining the pathological condition.

SUMMARY

1. A measuring instrument is described which enables the surgeon to determine the antero-posterior diameter of the vertebral canal during operation.

2. Developmental narrowness of the lumbar vertebral canal is shown to exist and to be caused by an abnormally short antero-posterior diameter.

3. In patients with a narrow, although not abnormally narrow, lumbar vertebral canal, slight deformities such as posterior spur formation or small disc protrusions may produce particular symptoms, which are interesting from a clinical as well as from a medico-legal point of view.

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A CHONDROMATOUS TUMOUR OF THE CALCANEUM

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Tumours of the long bones and of the talus and calcaneum often present difficulties in exact diagnosis and in treatment. These difficulties arise because it is often impossible to give a firm diagnosis of malignancy from examination of the first biopsy material, and conservative treatment is not uncommonly undertaken, which later is found to be inadequate.

The case here considered, a malignant chondromatous tumour of the calcaneum arising from a benign chondroma, is of interest because it occurred in one of the less common sites and because it clearly illustrates the above points. Geschickter and Copeland (1949) gave details of 113 benign chondromata in large bones, of which four were in the calcaneum: of eighty-three cases of chondromyxosarcoma following osteochondroma, three were calcaneal.

CASE REPORT

A furniture packer now aged sixty-one was first seen in 1946 on account of a painful heel. He recalled that in 1914 the wheel of a cart ran over his left heel. Apart from bruising

Fig. 1

Left calcaneum immediately before curettage and insertion of cancellous bone grafts.

there was little disability and the pain disappeared after a few weeks. He had no further trouble until 1939 when a falling window frame struck the same heel and cut it open. One stitch was inserted and the would healed rapidly without complications. Since that time he had occasional pain, but in 1946 it became much worse. He attended an orthopaedic clinic, where radiographs showed an area of rarefaction in the left calcaneum (Fig. 1). A diagnosis of chondroma was made. He was admitted to hospital and the cavity in the bone was exposed and curetted.

The material removed appeared macroscopically to be typical chondromatous tissue, and this was confirmed later by microscopy. Cancellous chips taken from the upper end of the tibia of the same leg were inserted into the cavity. Radiographs three months later

showed good sclerosis of the cavity (Fig. 2). Progress was satisfactory until 1949 when the patient had further pain in the left heel and noticed a swelling on the medial side of the left foot. A radiograph showed that there was a recurrence of the tumour on a more extensive scale (Fig. 3).

The left calcaneum was excised, because at that time the evidence of malignancy was not conclusive. A large tumour was found involving the bone and in places extending outside it. The tumour appeared encapsulated, and it did not appear to have invaded the soft tissues.

FIG. 2
Three months after curettage and grafting.

FIG. 3
Three years later. Radiograph shows extension of the tumour.

The wound healed normally and the patient walked well in a surgical boot. He returned to work eight months after the operation.

In 1953, four years after the calcaneum had been excised, it was noticed that the left heel was swollen and the soft-tissue shadow in the radiograph was more dense and enlarged. The swelling of the heel was explored. It was found to contain blood-stained fluid with sago-like particles in it. A below-knee amputation was carried out. The skin edges necrosed and the wound broke down. Dead tissue was excised and it was noticed that the healing tissue in the wound was not typical granulation tissue and a piece was sent for section. A small swelling was noticed also in the site of the donor area of the graft, where a radiograph revealed a translucent area (Fig. 4). The swelling was aspirated and material was removed resembling that withdrawn from the tumour of the heel shortly before amputation. An

FIG. 4

Metastasis in donor area of tibia, four years after removal of cancellous grafts.

above-knee amputation was carried out. The stump healed soundly. He has been fitted with an artificial limb and is walking well. So far there has been no evidence of a local recurrence or of pulmonary metastases.

Pathological findings—The following is a summary of the material examined.

The calcaneum (excised 1949)—The specimen consisted of a calcaneum to which was attached a mass of tumour tissue (Fig. 5). The articular surfaces appeared normal, but surrounding the posterior part of the bone and obscuring its outline was a mass of greyish-white material of cartilaginous consistency. The specimen was divided by vertical section, which showed soft white nodules of cartilaginous material surrounding the bone: on the under surface the cortex had been penetrated and the white cartilaginous material was occupying about a third of the medulla.

Microscopically the tumour was composed of rather cellular and not fully "adult" cartilage, and there was degeneration in the central parts (Fig. 6). The cartilaginous mass had the appearance of growing into the calcaneum and threading its way between the bone trabeculae, some of which were in process of absorption. The cartilage here was immature and some of it had a myxomatous appearance. There was no evidence of invasion of the small veins of the bone.

FIG. 5

Photograph of the divided calcaneum with chondromatous tumour.

Material removed from heel before amputation (1953)—The material consisted of a mass of jelly-like tissue which microscopically was composed of cartilage undergoing degeneration and often showing complete necrosis (Fig. 7).

Lower half of the leg and foot (amputated 1953)—The area previously occupied by the calcaneum was replaced by a mass of soft cartilaginous tissue (Fig. 8). Microscopically the cartilage varied much in appearance: some of it was of normal pattern, but much of it was softer with a poor matrix, and had a high cell content. It appeared to be actively growing.

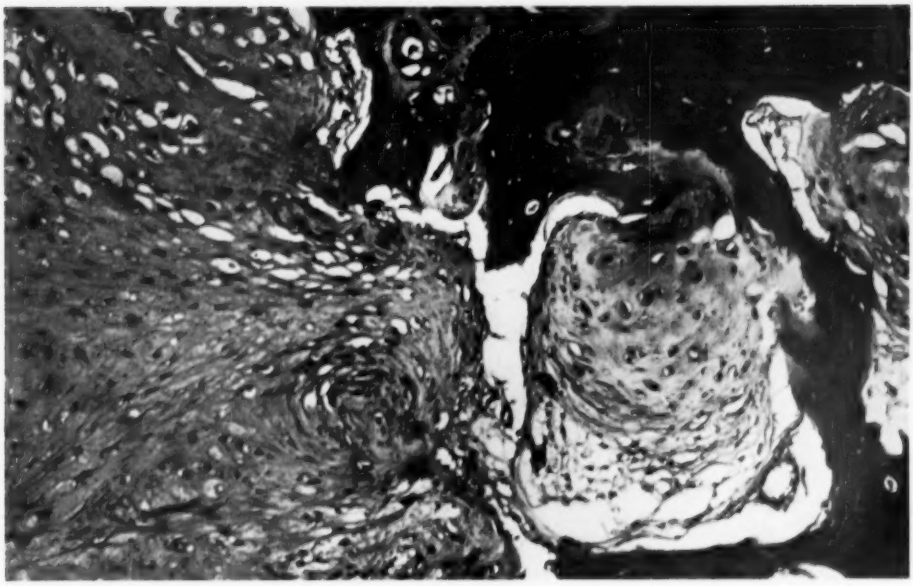

FIG. 6
Chondroma filling interstices of bone. (H. & E., ×80.)

FIG. 7
Degenerated cartilage evacuated from heel. (H. & E., ×80.)

Material taken from the tibial amputation stump and from a cyst-like swelling near the line of excision—Both of these specimens showed vascular material with a bluish chondroid matrix containing large numbers of spindle cells which were considered to be chondrosarcomatous, metastatic from the original lesion.

Specimen from part of the tibia from which material for grafting had been removed—In the space in the tibia from which bone for grafting had been removed there were several fairly well circumscribed vascular areas in which the capillaries were surrounded by spindle cells lying in a basophilic matrix (Figs. 9 and 10). In view of the antecedents of the case these areas were considered to be chondrosarcomatous.

DISCUSSION

The histological problem presented by this case is a common one. In spite of well-formed cartilage in the original tumour, the presence of some "plump" cells and the fact that the lesion was in the calcaneum led to an opinion that the tumour was potentially malignant.

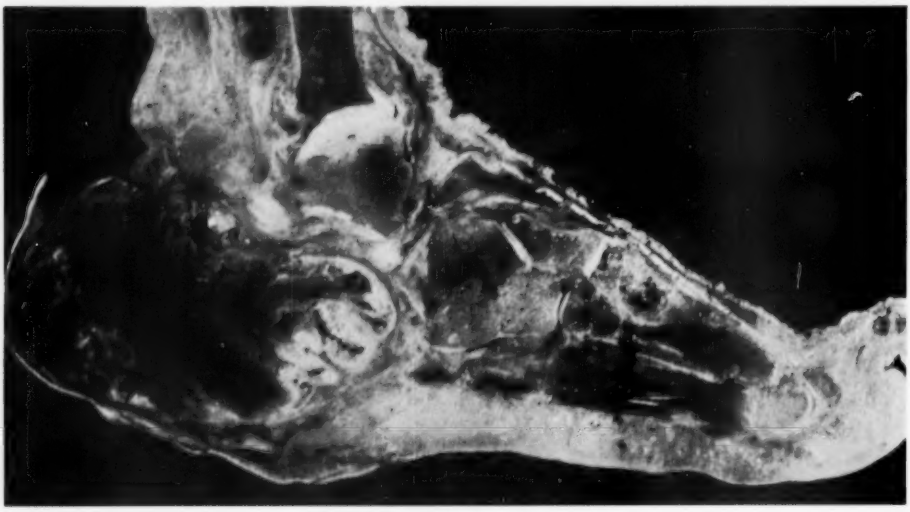

FIG. 8

Longitudinal section of foot after amputation.

It is indeed better to rely on the known behaviour of these tumours than on histological appearances unless, of course, the first biopsy shows unequivocal sarcoma. Local removal of the lesion is generally followed by repeated recurrences and not uncommonly the final result is a frank chondrosarcoma.

Geschickter and Copeland (1949) mentioned that although enchondromata of the small bones of the hands and feet may be curable by thorough curettage and cauterisation, this does not apply to the calcaneum or talus, and in these situations biopsy should be followed by radical resection because the tumours are easily transplanted. These observations are much to the point in the case under discussion. Figure 8 shows the tumour widely infiltrating the interspaces of the bone and extirpation by curettage could hardly hope to eradicate it completely.

The fact that a recurrence was found in the donor area in the tibia, although so far as is known separate instruments were used for this stage of the operation, illustrates the ease of transplantation. This risk could be avoided by removing the bone graft and closing the wound

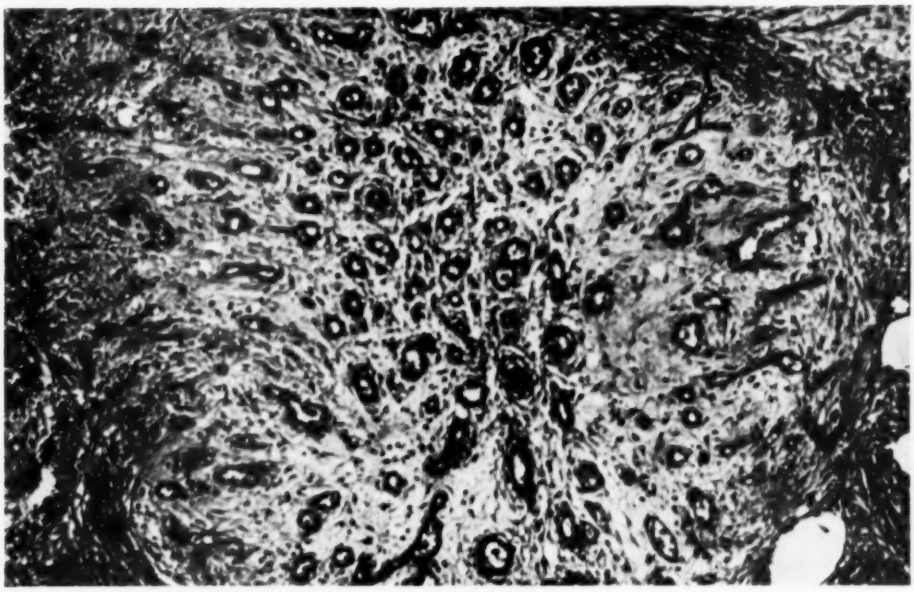

FIG. 9
Metastatic tumour from donor area in tibia. (H. & E., ×80.)

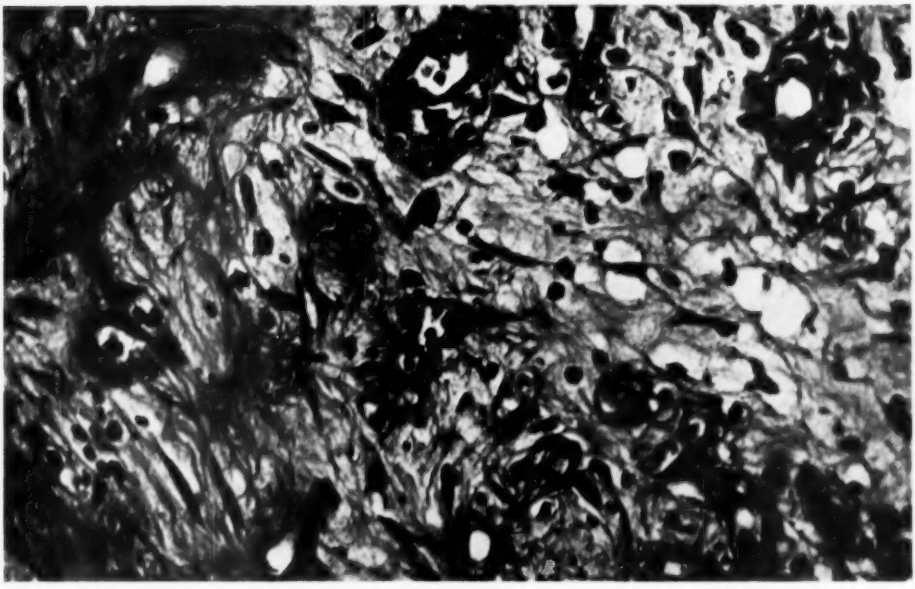

FIG. 10
Same as Figure 9 showing chondroid vascular matrix with large active cells. (H. & E., ×320.)

before making the incision for evacuating the tumour. Looking back on this case, it would have been better to amputate the foot immediately the diagnosis had been verified by biopsy. When doubt exists, amputation is the treatment of choice.

As with so many bone tumours, the role of trauma has to be considered but it is impossible to be dogmatic about the part, if any, which it played either in causing the original simple chondroma or its malignant transformation. The heel was injured twice, once in 1914 and again twenty-five years later. There is no evidence to show when the enchondroma of the calcaneum first appeared, but it was seen radiologically in 1946, seven years after the second injury.

Three current opinions on the relationship between trauma and bone tumours may be quoted. Willis (1953) wrote: " Trauma either plays no part at all or only a very infrequent one in the causation of bone tumours " and " the attitude for scientific pathologists is one of stringent scepticism." Bennett (1948), writing of osteogenic sarcoma, stated: " Many tumours are noted soon after trauma but it is usually impossible, in a given case, to prove an etiological relationship." Lichtenstein (1952) summed up his opinion as follows: " Altogether then, even in occasional cases in which there seems to be a significant factor of trauma, it cannot be accepted without some reservation. The natural tendency toward malignant transformation of osteochondroma is stronger in cases of multiple than in cases of solitary osteo-cartilaginous exostosis." In this case we must leave the matter as not proven, especially as the heel of all places is a part of the body that is normally subject to a considerable amount of trauma; nevertheless chondroma of the calcaneum is uncommon.

Although our patient is alive and well sixteen months after the final amputation and the lungs show no radiological evidence of metastases, the prognosis must still be guarded because pulmonary metastases have been known to develop from this type of lesion after several years.

SUMMARY

The characteristics of enchondromata of long bones are mentioned and the special features of a malignant chondroma of the calcaneum are described. The management of such a case is discussed.

We are indebted to Mr A. Reaney for the reproduction of the radiographs and to Mr C. O. Dyson for the photographs of the gross specimens and for the photomicrographs.

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THE USE OF LIVELY SPLINTS IN UPPER LIMB PARALYSIS

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A problem in the treatment of upper limb paralysis is to decide between reconstructive surgery and the provision of functional splints. Seddon (1952) stated: "There is no apparatus for the permanent control of the wrist, elbow or shoulder which approaches in usefulness what can be achieved by surgical intervention." Reconstructive surgery, using a combination of tendon transplants and joint stabilisation, has given remarkable results in the restoration of a functional arm (Barr 1949, Seddon 1952). There is, nevertheless, a growing tendency to use lively splints in certain types of paralysis. Capener (1946, 1949) has described a wide range of such splints for hand, wrist and elbow weakness, and there have been many reports in the literature describing new and improved lively splints (Highet 1942, Bunnell 1949, Stewart 1948, Irwin and Eyler 1951, Nangle 1951, Gazeley and Dunham 1952).

There are three main indications for the use of splints in upper limb paralysis. First, to prevent deformity, as for example in the maintenance of the limb in the position of function during the acute stage of poliomyelitis and the splinting of peripheral nerve injuries during the stage of regeneration. Second, to correct deformity—an example of this type is the use of stretch splints for tendon and muscle contracture. Third, to replace and encourage function that is permanently lost, or while recovery is proceeding.

Most of the splints described in the literature are designed primarily to prevent or correct deformity. Insufficient attention has been paid, in our opinion, to the design and development of lively splints to encourage function during the recovery stage or as a permenent replacement for lost muscle power.

This paper attempts to state the indications for their consideration in place of reconstructive surgery. The splints that have been found useful at this Centre are illustrated and described in detail.

ADVANTAGES OF LIVELY SPLINTS

We consider that lively splints offer certain advantages as a permanent or semi-permanent solution rather than surgery. Firstly, they can be made quickly and after a short trial period the patient can return to employment. In over half the cases in this series it was essential for the patient to start training or to return to employment as quickly as possible. Many could not afford the long periods in hospital and rehabilitation units necessitated by a comprehensive reconstructive programme, which may not be completed until many months or years after the original disease or injury. The longer a patient is off work under treatment, the more difficult it is for him to return to normal life and working conditions.

Secondly, lively splints by their nature offer movement, where reconstructive surgery can sometimes offer only stability. An example of this is when there is paralysis of biceps, and flexion of the elbow is required but the pectoralis major is paralysed or too weak to be usefully transplanted. In our opinion a bone block to the elbow is not so efficient functionally as the lively splint described in this paper. Although lively splints may be somewhat unwieldy, some patients prefer the flexibility offered to the fixation produced by bone blocks and arthrodesis.

Thirdly, it is not unknown for reconstructive surgery to turn out rather differently from what was originally intended. Apart from technical factors, the after-treatment and the patient's full co-operation are equally important for the success of an operation. In one case

a lively splint added to the usefulness of reconstructive surgery. This patient had almost complete paralysis of the right arm. A bone block was performed to give him abduction of the thumb. The provision of an opponens splint enabled him to write more efficiently. It is sometimes difficult for patients to appreciate exactly what the results of reconstruction will be. We have found that a splint made to imitate the results of such an operation and worn for a period of two to three weeks will give the patient and the doctor a fairly exact idea of the likely end result. In two of our patients opponens splints were worn to imitate the action of a transplant, and as a result the patients decided on operation, which was successfully carried out.

Fourthly, the patient himself may be reluctant to accept such a radical measure as reconstruction. Six of the patients with severe paralysis were convinced that they would get further recovery despite the clinical and electrical evidence to the contrary. Many more patients were much happier to accept lively splintage at least for the time being because, as one patient put it, " one can always have one's joint fixed, but once it is done it is done for good."

Finally, the splints are easy to make, require only the simplest materials, and are cheap. The adjustable elbow splint, for example, costs about three shillings and the opponens splint only sixpence to make. There is undoubtedly a place for lively splintage during the recovery period or while nerve regeneration is awaited. The splints offer as near normal function as possible and thus help to lay down correct movement patterns. They can also provide means for the actual rehabilitation of muscle groups. The biceps spring provides resistance against which the weak triceps can work.

DESCRIPTION OF SPLINTS

During the last eighteen months splints have been made for twenty-six patients with various types of upper limb paralysis. All the splints have been made in the occupational therapy department with the simplest of materials and at small expense. Some of the splints to be described may be original, but most are modifications of conventional types that we have found to be helpful and sometimes essential in providing efficient function.

The conditions for which splints were provided fall into four groups: 1) disabilities involving elbow alone; 2) disabilities involving elbow, wrist and hand; 3) disabilities involving wrist and hand; 4) disabilities involving hand alone. The causative lesions were: brachial plexus lesions 4; poliomyelitis 8; peripheral nerve injuries—ulnar 4, median 3, radial 5; arthroplasty of elbow 2.

SPLINTS FOR DISABILITIES INVOLVING THE ELBOW ALONE

Nine patients had weakness or paralysis confined to the elbow. The splints to be described were also used for patients who had associated wrist or hand weakness, but these will be discussed in a later section.

In two patients, one with arthroplasty of the elbow and one whose olecranon had been excised, the disability was severe weakness of the triceps. In these two patients the splint (Fig. 1) was eventually discarded when the triceps had regained control over the elbow. This splint was also used in two other patients with poliomyelitis who had weakness of both flexion and extension of the elbow. The primary aim was to assist them in function during retraining for their employment, but incidentally it also provided a convenient means of giving resistance to the weak triceps for its rehabilitation.

During treatment it is often necessary to support the arm in a sling. A sling has been devised which provides complete support for the shoulder but avoids putting extra weight on the cervical region (Fig. 2).

When triceps weakness or paralysis is permanent in the presence of normal flexion of the elbow, a different type of splint is required (Fig. 3). This was designed for a patient

with severe paralysis from poliomyelitis. Both legs were completely paralysed and the only useful movements in the arms were flexion of the elbows and a pincer movement of the right

FIG. 1

Elbow splint for weak triceps. The strength of the spring is graded according to the strength of the triceps, allowing the elbow to obtain full extension. As the spring requires maximal power to effect full extension, constant use is an efficient form of triceps re-education.

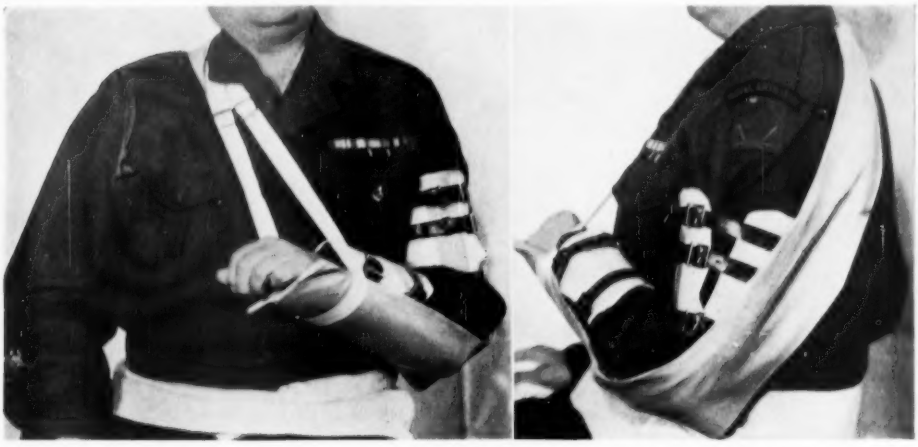

FIG. 2

Shoulder sling. A piece of canvas 7 inches wide and 64 inches long has an envelope piece folded back, the length of which is from the elbow to the tip of the fingers. Into this is inserted a piece of curved aluminium alloy. At the other end are two buckles at either side of the hand piece, into which fit two straps from the shoulder piece. The sling is adjustable and is useful for control of oedema of the hand and forearm.

finger and thumb. The absence of triceps made it impossible for him to manipulate his wheel chair. The splint allowed good extension of the right elbow and enabled him to move his chair unaided.

Two patients with poliomyelitis suffered from complete paralysis of flexion of the elbow and weakness of triceps (grade 3 on Medical Research Council scale). The splint for these patients is similar to the one just described, except that the spring is attached to the anterior aspect of the bars and is in relation to the weight of the forearm and the power of the triceps

FIG. 3

Elbow extension splint. A duralumin bar with an elbow hinge is fitted to the lateral aspect of each moulded leather cuff. A nylon thread attached to the bottom of the spring runs down in a groove behind the hinge to the hook on the forearm cuff.

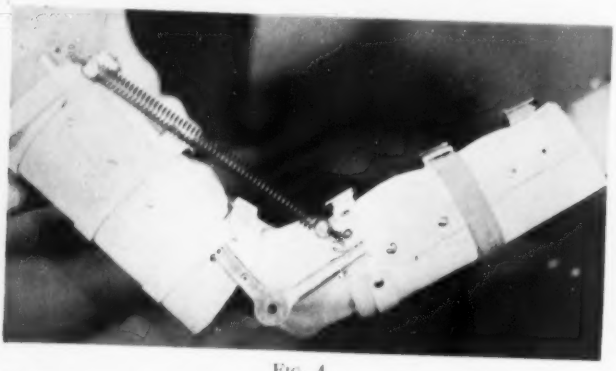

FIG. 4

Elbow flexion splint. This splint is the same as that illustrated in Figure 3 except that the hooks are attached to the front of the bars. The power of the spring is in relation to the weight of the forearm and the power of the triceps.

(Fig. 4). The object of the splint is to bring the elbow into mid-flexion, and it enabled one of the patients confined to a wheel chair to move his chair unaided. The splint also offers resistance to the weak triceps, thereby aiding its rehabilitation.

A patient with a lesion of the brachial plexus involving C.5–6 had paralysis of abduction of the arm and flexion and supination of the elbow. A programme of reconstructive surgery was refused. A splint was made to provide supination and flexion of the elbow. The splint

was worn inside the jacket and the springs outside. It was constructed with the patient's job as telephone operator in mind and allowed him to hold the earpiece in his left hand (Fig. 5). (A new type of spring that can be worn inside the jacket will be described later.)

A patient with a total brachial plexus palsy underwent extensive reconstructive surgery,

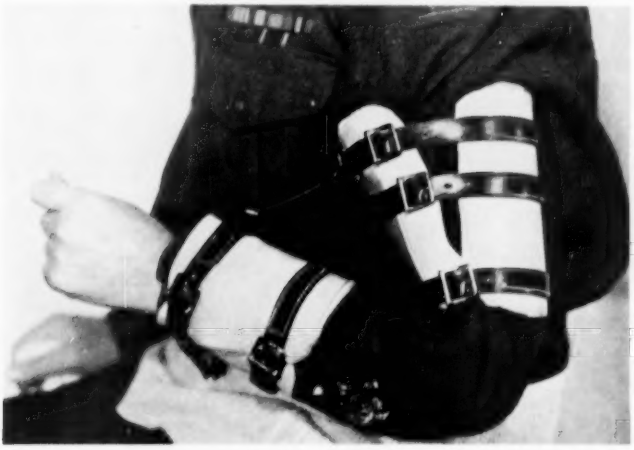

FIG. 5

Lively elbow flexion splint. Three springs are provided, for the outer third, the middle third and the inner third of flexion respectively. The splint is worn inside the jacket and the springs outside. The splint was constructed with the patient's job as telephone operator in mind and the short spring allowed him to operate the earpiece in his left hand.

FIG. 6

Adjustable elbow splint. This splint is controlled by a leather strap passed from the upper arm piece through a roller pulley to a buckle on the forearm piece. The splint allows the patient to fix his elbow in any position desired.

including arthrodesis of the shoulder and wrist, bone blocks to the elbow and thumb, and tenodesis of the fingers. Unfortunately the bone block at the elbow was absorbed and the elbow again dropped into full extension. From the point of view of his work it was important that he should have some means of holding his elbow flexed. A splint was made to allow the patient to fix his elbow in any position he desired (Fig. 6).

FIG. 7

Adjustable elbow splint with lively wrist extension. The extension consists of two bars with a hinge at the wrist. A transverse padded bar just proximal to the metacarpal heads supports the hand. The strength of the spring is graded according to the strength of the wrist flexors.

FIG. 8

Adaption to splint to prevent shoulder subluxation. The adaption consists of a padded kidney-shaped piece of hide to which is added two straps. These straps fix on to buckles on the upper part of the elbow splint on the medial side, one in front and one behind. A supporting fabric strap passes round the chest from the back of the shoulder piece underneath the opposite arm.

SPLINTS FOR DISABILITIES INVOLVING ELBOW, WRIST AND HAND

Four patients had paralysis of the muscles controlling the elbow, the wrist, and extension of the metacarpo-phalangeal joints. Flexion of the fingers and intrinsic action of the thumb and fingers were powerful. The splints made for these patients included the elbow and wrist in one piece. The splint provided means of holding the elbow in any desired position and allowing the wrist to be brought into extension (Fig. 7).

Patients with severe paralysis of the shoulder muscles in addition to weakness of the elbow often show partial subluxation of the shoulder. This is not only painful but a serious

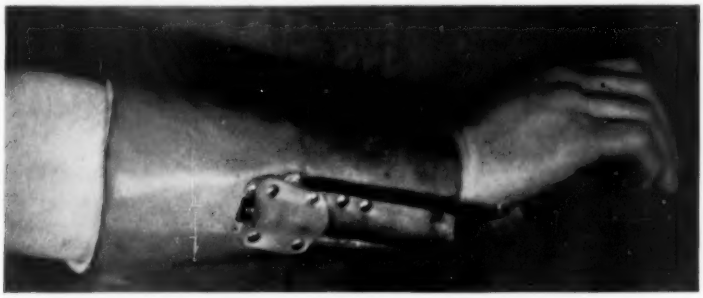

FIG. 9

Radial nerve splint. The splint is the same as the extension shown in Figure 7 except that the wrist piece extends just beyond the metacarpo-phalangeal joints and there is a metal roller piece on the transverse bar opposite the fifth metacarpo-phalangeal joint. This makes for smooth flexion of the fingers.

FIG. 10

Lively ulnar nerve splint. A Perspex bar rests on the back of the hand just proximal to the bases of the fingers. A ¼-inch dowelling bar is attached either side across the palm. A bar of Perspex fits across the middle of the proximal phalanges dorsally and the ends are attached to the hinges of the dowel by elastics or small springs. (This patient is also wearing an opponens splint for median nerve palsy.)

drawback to function. To combat this a special adaption has been fitted to the elbow (Fig. 8). This successfully prevents subluxation and is virtually no extra weight to the splint. It also provides a counter pull at the elbow against the forearm weight.

DISABILITIES INVOLVING THE WRIST AND HAND

Five patients had paralysis of extensors of the wrist, fingers and thumb. In three cases the paralysis was caused by injury to the radial nerve; all recovered fully. One was due to a gunshot injury of the brachial plexus which showed no recovery. The other was due to a posterior interosseous palsy caused by a blow with a hockey ball. The splint used in these patients is illustrated in Figure 9.

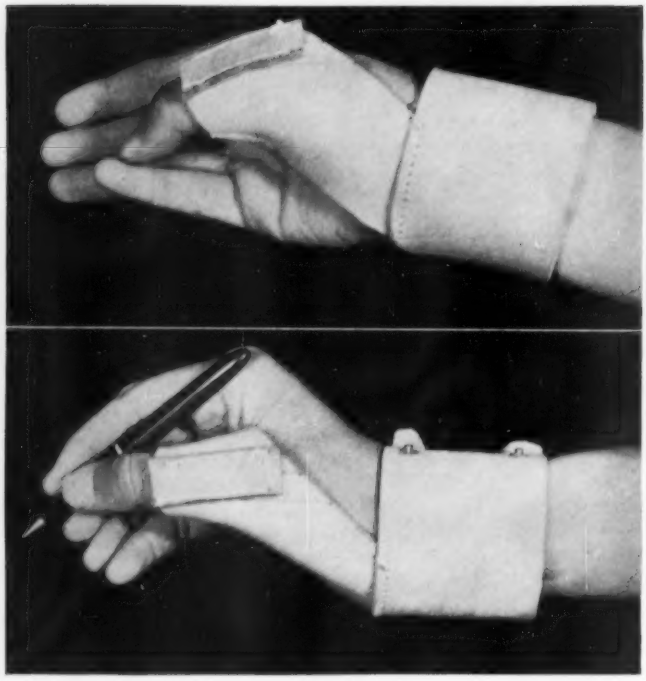

FIG. 11

Opponens splint. A piece of flexible rubber is moulded to enclose the proximal phalanx and to lie across the thenar eminence in the line of action of the opponens muscle: this is attached to a cuff encircling the wrist. A strip of stiff hide approximately ¾ inch wide, attached to the rubber, lies across the metacarpo-phalangeal joint. If full stability is not obtained by this means, a narrow strip of metal is used instead of the hide.

FIG. 12

Thumb stabilising splint. The splint consists of a moulded leather piece on the extensor aspect of the metacarpo-phalangeal joint and the metacarpal, encroaching slightly on the palmar aspect of the thumb and completely encircling the proximal phalanx. A moulded piece of Perspex is fitted over the extensor aspect of the metacarpo-phalangeal joint and the first metacarpal to give stability for writing.

DISABILITIES INVOLVING THE HAND ALONE

Ulnar nerve lesions—Lively splints have been made for five patients with ulnar nerve lesions. This splint prevents deformity at the metacarpo-phalangeal joints and allows full function of the hand, which is not permitted by the "knuckleduster" type of splint. This splint is

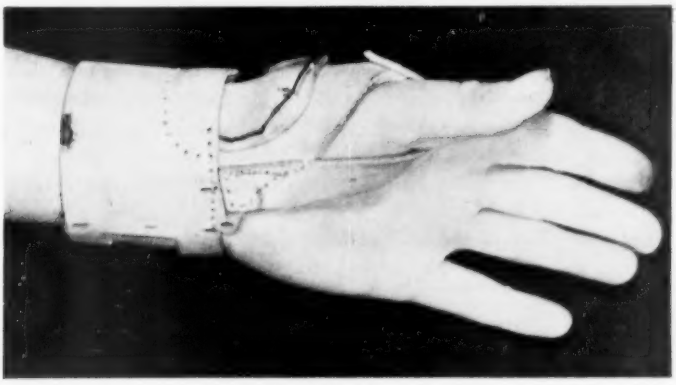

FIG. 13

Splint to control subluxation of the carpo-metacarpal joint of the thumb. This splint is like that shown in Figure 12, but the moulded piece of Perspex is stitched to the leather piece with a slight upward pressure over the extensor aspect of the carpo-metacarpal joint, encroaching slightly on its palmar aspect.

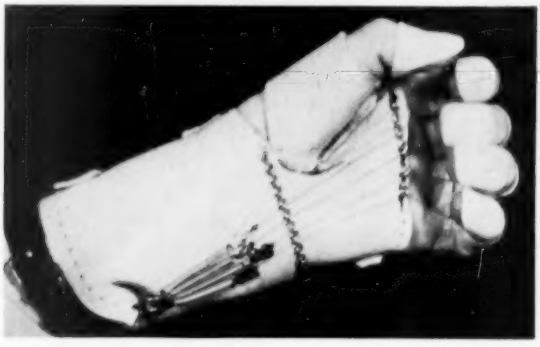

FIG. 14

Interim splint for paralysed finger flexors. Plastic rings are fitted over each phalanx, the terminal ring covering the nail but leaving the pad of the finger free. Each ring has a tiny horizontal metal bar incorporated on the palmar surface. The thumb is fitted with an opponens splint incorporated into the hand piece, and it has a terminal ring cap only. Nylon is attached to the terminal ring of each digit and passes under each bar and then through fine chain rings in front of the metacarpo-phalangeal joints and at the first wrist crease, to hook into springs attached five inches proximal to the wrist. Experience has proved that the terminal cap should extend a little farther over than is shown in the photograph.

useful during the stage when recovery of the nerve is awaited, but it can also be used as a permanent splint should recovery not occur. One patient using the splint for a permanent brachial plexus paralysis was able successfully to take a course in paint spraying. The splint is illustrated in Figure 10.

Weakness of the thenar muscles—Splints have been made for six patients with paralysis of opposition—three with median nerve lesions and three with poliomyelitis. The aim of the

splint is to abduct, rotate and control the thumb at the metacarpo-phalangeal joint (Fig. 11). All six patients using this splint were able to write with the pen correctly gripped and suffered no restriction of thumb movement. In two patients weakness of the thenar muscles required a stabilising splint without the need for rotation. One of these patients had weakness of the flexor pollicis brevis from poliomyelitis and the splint used is illustrated in Figure 12. The other patient suffered from weakness of all the thenar muscles with recurrent subluxation of the carpo-metacarpal joint (Fig. 13).

Paralysis of all finger flexors—A patient with paralysis of all the finger flexors from poliomyelitis was fitted with the splint shown in Figure 14. The splint restored a reasonable grasp.

SUMMARY

1. The indications for the use of lively splints in upper limb paralysis instead of reconstructive surgery are discussed.

2. Examples of lively splints used for the elbow, wrist and hand are described and illustrated.

We wish to thank Mr H. Osmond-Clarke, C.B.E., for his encouragement and valuable advice in the preparation of this paper. We wish to acknowledge the help of the workshops at the Medical Rehabilitation Unit, Royal Air Force, Collaton Cross, in making the metal hinges incorporated in some of the splints. We wish to thank also the Director General Medical Services, Royal Air Force, for permission to publish this paper.

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SACRAL EXTRADURAL CYST: AN UNCOMMON CAUSE OF LOW BACK PAIN

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The diagnosis of low back pain may be difficult, particularly if physical signs are absent or unusual. The following case illustrates an unusual cause, and shows some of the features and signs that may be expected when an extradural cyst occurs in the sacral region.

CASE REPORT

A merchant aged thirty-one years, referred by Mr Eric Davies, of Calcutta, had four years previously begun to suffer from an ache in the lower part of his back. This was

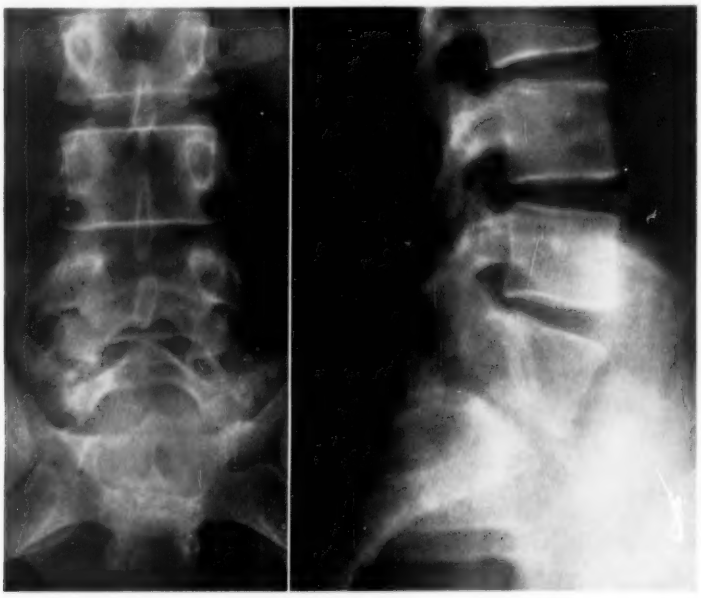

FIG. 1

Radiographs showing erosion of the posterior aspects of the bodies of L.6 and S.1, 2 and 3, with an ovoid expansion of the sacral canal. The ala of the sacrum is thinned and bowed backwards. The left pedicle of L.6 is eroded.

aggravated by bending but unaffected by coughing or straining. There was no history of injury. During the next three years he was troubled, at intervals of a few weeks, by attacks of backache. At the end of this time he suffered an additional sharp pain in the right sacro-iliac region which lasted for ten days. Thereafter he was free from symptoms until two months before his admission, when he had a further attack lasting only one day. A radiograph taken at this time showed an ovoid expansion of the vertebral canal (7·0 × 4·5 centimetres) in the lumbo-sacral region. This expansion had resulted chiefly from scalloping of the posterior aspects of the bodies of the upper three sacral vertebrae and of the lowest lumbar vertebra (of which there were six). In addition the pedicle of the sixth lumbar vertebra on the left side was eroded, and the ala of the sacrum was thinned and bowed backwards (Fig. 1).

These findings led to his admission to this unit. He was by then free from symptoms, and there were no abnormal findings on clinical examination. Lumbar puncture showed normal cerebrospinal fluid and pressure responses. Lumbar myelography showed that the lower part of the theca was attenuated and displaced backwards and to the right within the expanded sacral canal. Contrast medium could be seen to drip through a small orifice in the left side of the theca into a cystic cavity occupying the area of bone expansion (Fig. 2). Further radiographs taken two weeks later showed that all the contrast substance had entered the cyst, within which it moved freely (Fig. 3). These findings led to a provisional diagnosis of sacral extradural cyst. In view of the likelihood of recurrence of the patient's symptoms, and because of the possibility of sphincter paralysis recorded in comparable cases in the literature (Tarlov 1953), an exploration of the sacral region was decided upon.

Operation—The laminae of the fifth and sixth lumbar vertebrae and the thinned ala of the upper three sacral vertebrae were exposed by a posterior midline incision and removed

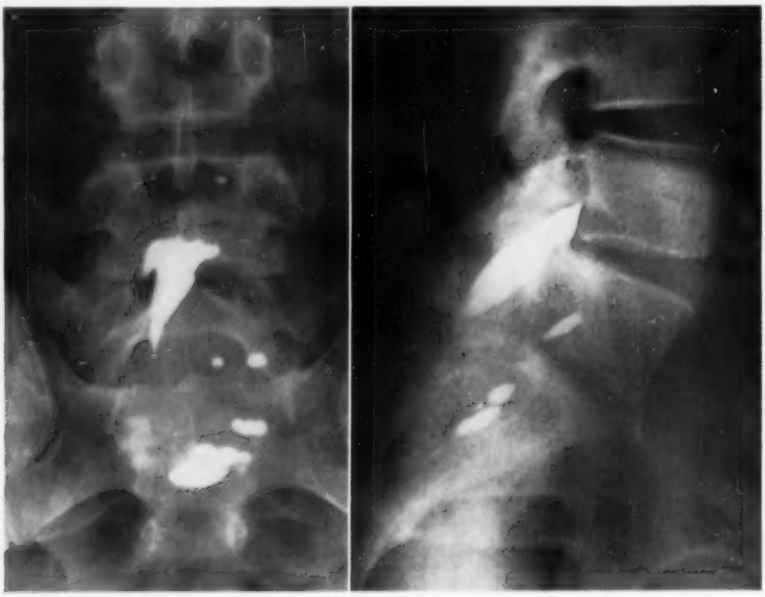

FIG. 2

Initial myelogram with the patient erect, showing displacement of the lower end of the theca to the right. Below this a few drops of contrast medium have collected within the extradural cyst.

(Mr M. A. Falconer). A sac consisting of a thin transparent membrane containing cerebrospinal fluid was found, occupying the greater part of the sacral canal. This sac was traversed by the second and third left sacral nerves, which were normal in appearance, and it had displaced the theca to the right and backwards. At the origin of the dural root sleeve of the second left sacral nerve a small pouch of arachnoid had herniated into the cyst and cerebrospinal fluid could be seen dripping into the cyst cavity from an opening in this pouch. The valvular nature of the aperture had evidently prevented the cerebrospinal fluid from returning to the subarachnoid space; fluid had therefore gradually accumulated and given rise to a false sac which had eroded and expanded the adjacent bone. In order to repair the herniation and close the leak in the arachnoid, the theca was opened with a posterior incision, and the arachnoidal sheath around the second left sacral nerve roots was divided where it entered

the dural sleeve. A collar of fascia was sutured around the roots at this point, thus sealing the opening in the dura. The posterior thecal incision was then closed. Histological examination of the cyst wall (Dr D. Naidoo) showed it to be composed of collagenous fibrous tissue without any sign of nerve fibres or ganglion cells.

Progress—Recovery from the operation was satisfactory. Post-operative myelography showed that contrast medium would no longer pass out from the spinal theca, and that the displacement of its lower end was now almost corrected. A year later the patient wrote that he was free from back pain, although he still experienced occasional discomfort in the back of his legs on strenuous exercise.

DISCUSSION

Pain similar to that described by this patient may be associated with intervertebral disc lesions, cauda equina tumours, spondylolisthesis, and tuberculosis of the sacro-iliac region,

FIG. 3

Myelogram taken two weeks later with the patient in the head down position. Contrast medium is now entirely within the cyst and will not return to the spinal theca.

as well as by sacral cysts of perineurial or extradural type. The erosion of bone seen in the plain radiographs made disc protrusion alone an unlikely cause of his symptoms, and there was no sign of spina bifida. It required myelography to elucidate the problem further; this demonstrated the cystic nature of the lesion and showed that the expansion and erosion of bone were not due to pressure by a neoplasm.

The pathological nature of perineurial and extradural sacral cysts requires clarification. The perineurial sacral cysts described by Tarlov (1938, 1953) occur at the junction of the posterior nerve root with the dorsal root ganglion, and arise in the space between perineurium and endoneurium; they may be nearly solid and may contain nerve fibres and ganglion cells. Communication with the subarachnoid space is never very free, and is usually not present. Erosion of bone and separation of the vertebral pedicles rarely occurs (Fig. 4).

In our case the cyst was of the extradural type. Such cysts were first comprehensively reviewed by Elsberg, Dyke and Brewer (1934), who found that most of those reported had occurred in the mid-thoracic region in patients under twenty years of age; Cloward and Bucy (1937) showed that the condition was frequently associated with adolescent kyphosis (Scheuermann's disease). No sacral example was described until the paper by Schreiber and Haddad (1951) appeared; since then three other sacral examples have been reported by Strully and Heiser (1954).

Extradural cysts arise from a diverticulum of arachnoid which has herniated through a dural defect. All the sacral examples reported have communicated freely with the subarachnoid space, though with cysts at other levels this communication has usually been obliterated. The wall consists of fibrous tissue and may or may not be lined with arachnoid-like cells. Sometimes, as in the case described, it may be surrounded by a much larger false sac produced by leakage of cerebrospinal fluid through a valvular opening in the true sac.

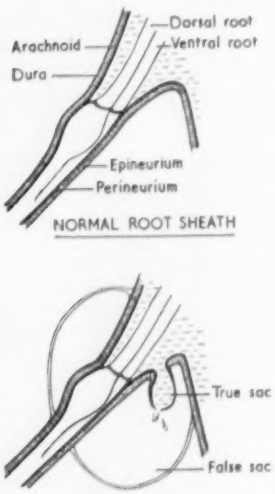

FIG. 4
Diagrams of extradural and perineurial cysts.

The origin of extradural cysts may be spontaneous or post-traumatic. The spontaneous variety owe their existence to a congenital failure of fusion of mesenchymal structures surrounding the neural tube, in which case they are a form of meningocoele, or they may arise (as in our case) from a defect in the dura mater adjacent to or at the origin of a root sheath. Post-traumatic extradural cysts were first reported by Hyndman and Gerber (1946), who described cervical and lumbar examples following spinal operations. One of the sacral extradural cysts reported by Strully and Heiser (1954) was of this type; their other two cases were spontaneous in origin, as was our own (Fig. 4).

There are no constant clinical features which allow sacral extradural cysts to be differentiated from perineurial sacral cysts or from prolapsed nucleus pulposus. The radiological appearances may, however, be of the utmost value in distinguishing them, since bone is generally expanded and eroded only by the extradural variety. Myelography amplifies the information, and if the contrast medium does not readily enter the cyst, late films (after at least twenty-four hours) should also be taken because its passage may be slow. Perineurial cysts do not usually communicate with the subarachnoid space sufficiently freely for them to be filled by the contrast substance, although small amounts may occasionally be seen to enter (Taheri, Riemenschneider and Ecker 1952, Strully and Heiser 1954). The presence of contrast medium within a sac, in conjunction with erosion of bone or widening of the vertebral canal, is strong evidence in favour of an extradural cyst. The case described showed all these features.

Most of the reported cases of extradural cyst have shown progressively increasing disability, despite remissions. Furthermore, if the condition is allowed to progress, sphincter disturbance from compression of the cauda equina is likely to follow, as has been described in conjunction with the perineurial variety (Tarlov 1953). Operation should therefore always be considered since evacuation of the false sac, if one is present, followed by removal or reduction of the arachnoidal sac and repair of the dural defect, has usually been followed by relief of symptoms.

SUMMARY

1. A case of low back pain due to a sacral extradural cyst is reported. Radiographs of the sacrum showed an ovoid expansion of the sacral canal. Myelography and exploratory laminectomy revealed an extradural cyst associated with a defect in the dural root sleeve surrounding the second left sacral roots. Relief of symptoms followed evacuation of the cyst and repair of the defect.

2. The differential diagnosis of the condition, the varieties of extradural cyst, and the features which distinguish them from perineurial cysts are described.

This patient was under the care of Mr Murray A. Falconer, Director of the Guy's-Maudsley Neurosurgical Unit, to whom I am indebted for permission to publish this report.

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OSTEOGENIC FIBROMA OF BONE

Report of a Case

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From Raigmore Hospital, Inverness

In this paper we shall record a case of a tumour-like process affecting the vertebral column which, in its clinical, radiological and pathological characters resembles the lesion Lichtenstein (1952) designated osteogenic fibroma of bone. This is an uncommon condition which is liable to give rise to difficulties in diagnosis. Osteogenic fibroma is one type of a group of benign tumour-like processes of bone which are composed of essentially the same constituents, osteoid tissue and more or less calcified atypical bone in a matrix of highly vascular osteoblastic connective tissue. The members of the group present certain differences in respect of growth capacity and the reaction of adjacent tissues, and of their clinical and radiological characters (Jaffe 1935, Jaffe and Lichtenstein 1940, Lichtenstein 1952, Golding 1954, Golding and Sissons 1954). The group includes the lesion known as osteoid osteoma (Jaffe 1935, Jaffe and Lichtenstein 1940, Golding 1954), osteogenic fibroma of bone (Lichtenstein 1952, Golding and Sissons 1954), and certain rarer tumours occurring in children and young adults which may become more aggressive in the course of their development without becoming frankly malignant (Lichtenstein 1952).

CASE REPORT

In 1948 a boy of thirteen was admitted to the hospital with paraplegia of recent onset, and complaining of pain in his back. About six months previously he had fallen from the roof of a shed, but was not aware that he had injured his back, and did not complain of pain until three months later. Two days before admission his legs suddenly became weak, and this weakness became rapidly worse.

On examination he was found to have a tender lower thoracic kyphos with soft-tissue thickening on both sides, especially the right. Both lower limbs were paralysed and there was loss of sphincter control, but no loss of sensation was demonstrated. Radiographs of the spine (Fig. 1) showed a semi-translucent area in the right side of the ninth thoracic vertebra, with a suggestion of new bone formation and expansion laterally. The lesion was apparently confined to the lamina, pedicle and transverse process, without involving the vertebral body. The chest was clear. The Wassermann reaction was negative, and the only abnormality found in the blood was an erythrocyte sedimentation rate of 43 millimetres in the first hour (Wintrobe's method). This was ascribed to a urinary infection, which was already present.

Three days after admission sensory impairment was noted in the lower limbs, and this rapidly progressed in the space of two days to complete sensory loss below the level of the twelfth thoracic segment.

Operation—Laminectomy was undertaken. While the muscles were being stripped from the spinous processes and laminae, excessive bleeding was encountered on the right side, and a soft haemorrhagic tumour-like mass was found to be protruding between the laminae of the eighth and ninth thoracic vertebrae. Closer inspection showed actual invasion of these laminae. Laminectomy was proceeded with and as much of the tumour as possible was removed. The dura, which was seriously compressed but not actually invaded so far as could be seen, was not opened. The origin of the tumour was not ascertained, but it was thought

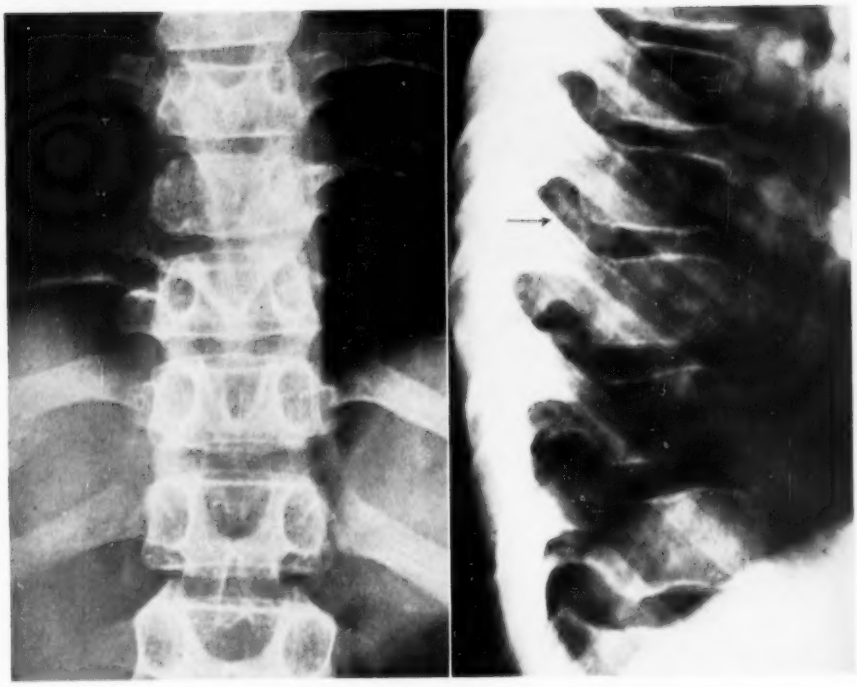

FIG. 1

Radiograph on admission showing lesion in right side of the ninth thoracic vertebra. The vertebral body is not involved. The arrow points to a calcified mass above pedicle of T.9.

FIG. 2

FIG. 3

Figure 2—Radiograph ten weeks after laminectomy, showing expansion and ossification of tumour.
Figure 3—Radiograph two and a half years later.

that it possibly arose in the pedicle, and from its naked-eye appearance the tumour was considered to be almost certainly malignant. After operation the boy was nursed on a plaster bed, and the spinal lesion was treated by x-ray therapy.

Progress—Ten weeks after operation radiographs showed lateral expansion of the tumour and well marked ossification throughout (Fig. 2). The clinical condition remained unchanged, and with the gloomiest of prognosis he was allowed home, where his mother continued to nurse him on the plaster bed.

After a few months he began to show signs of recovery of cord function, and as his general condition continued to be good he was readmitted to hospital. At that time both lower limbs were very spastic, but he had regained a little voluntary control of feet and knees, sensory loss had diminished in extent and degree, and he was no longer incontinent of faeces. Radiographs now showed the spinal lesion to be sclerosed with sharply outlined borders, and bulging quite far to the right of the vertebral body.

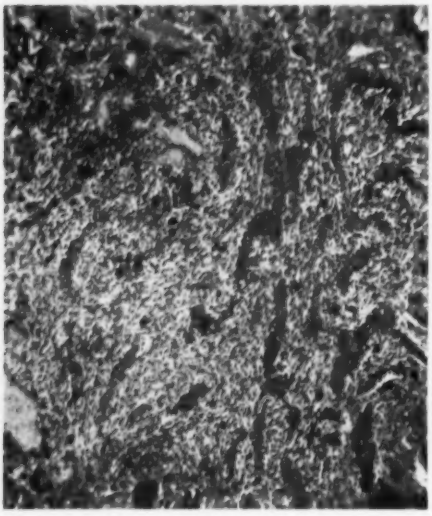

FIG. 4

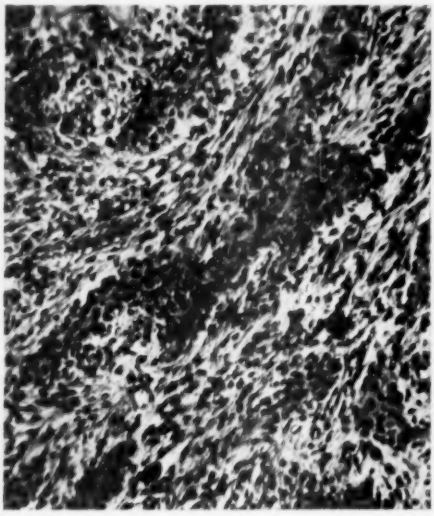

FIG. 5

Figure 4—Cellular connective tissue showing active differentiation into irregular bands of osteoid tissue. (×55.) Figure 5—Cellular connective tissue showing differentiation into irregular bands of osteoid tissue. No notable degree of cytological irregularity. (×140.)

After a second course of radiotherapy to the spine, every effort was made to rehabilitate him so far as possible by physiotherapy, neurectomies and braces, and after three years he was able to walk with elbow crutches and without calipers. Radiographs showed progressive ossification of the spinal lesion (Fig. 3). When he was last seen seven years after the onset of symptoms he remained in good general health, and there was no clinical or radiological evidence of metastases.

Pathological examination of excised material—The material consisted of tumour tissue and pieces of adjacent bone. Macroscopically, the tumour tissue was soft, haemorrhagic and "gritty" to the touch. A little soft tissue, similar in character, was attached to the pieces of bone.

Histological examination—Tumour tissue: this consisted of highly cellular and vascular connective tissue mixed with recent blood-clot. In many parts of this cellular tissue active differentiation into irregular areas and bands of osteoid tissue was in progress (Figs. 4 and 5). In other parts the cellularity was more marked and differentiation much more irregular, and

in such areas there was often a deposition of calcium salts on highly irregular spicules of bone (Fig. 6). Scattered throughout the tissue osteoclasts were present in moderate numbers (Figs. 4, 8 and 9). Mitoses were present but they were not numerous.

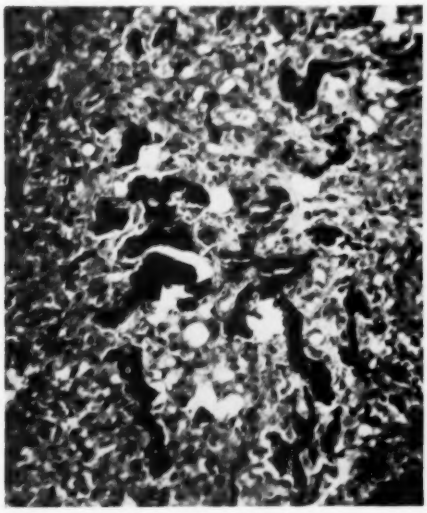

FIG. 6

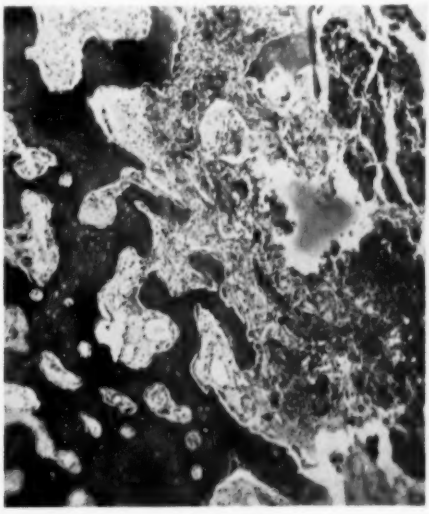

FIG. 7

Figure 6—Markedly cellular and atypical tissue containing irregular spicules of calcified bone. (× 140.)
Figure 7—Normal bone (*left*) bounding tumour tissue (*right*). (× 55.)

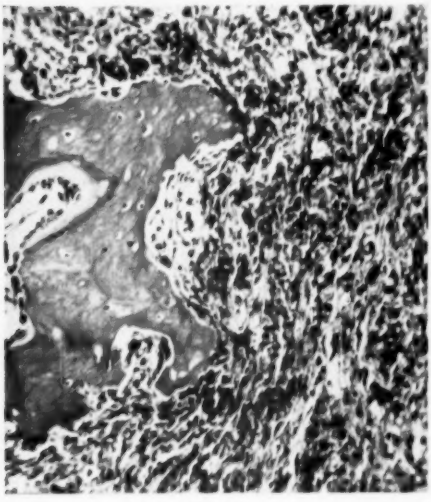

FIG. 8

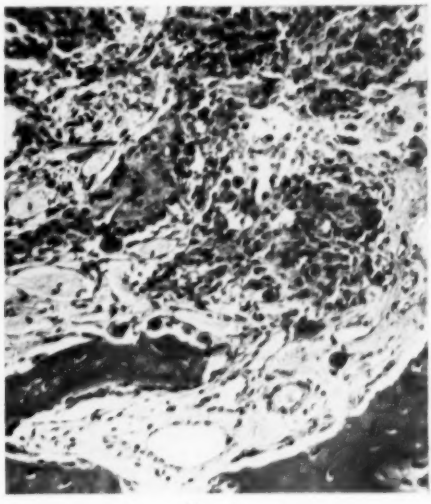

FIG. 9

Tumour tissue infiltrating cancellous spaces of normal bone. (× 140.)

Pieces of adjacent bone: this consisted chiefly of normal bone (Fig. 7), some of which was newly formed. At parts of the inner surface of the bone and extending into the cancellous spaces, there were plaques of cellular osteoblastic tissue similar in character to the tumour tissue (Figs. 8 and 9).

DISCUSSION

At the time of the surgical exploration diagnosis in this case was difficult. At operation the lesion appeared to be a tumour invading bone and probably malignant. Microscopic examination of the material suggested either an osteogenic sarcoma or active and disorderly osseous granulation tissue. Although there was clear evidence of invasion of bone, the stromal cells did not exhibit the degree of cytological irregularity to be expected in osteogenic sarcoma, and mitoses were not numerous. On the other hand, there was no apparent explanation of the presence of osseous granulation tissue. From the subsequent history of the patient it seems clear that the lesion was not an osteogenic sarcoma. The radiographic appearances did not suggest giant-cell tumour of bone, and the histology of the biopsy material was quite unlike giant-cell tumour.

The diagnosis of osteogenic fibroma in this case depends on the absence of any discoverable lesion to explain the presence of osseous granulation tissue, on the clinical course which excludes osteogenic sarcoma, and on the similarity of the clinical, radiographic and pathological features to those in other recorded cases of osteogenic fibroma (Lichtenstein 1952, Golding and Sissons 1954).

As indicated above, osteogenic fibroma of bone is one type of a group of benign osteoblastic tumour-like processes of bone (Lichtenstein 1952). The scope and status of the whole group, and the relationship, if any, between the members of the group are not yet clearly defined.

Osteoid osteoma was formerly regarded as a chronic inflammatory process of bone, and still is so regarded by some observers (Brailsford 1942). Jaffe (1935), who made a careful study of the lesion, concluded that it is a benign tumour of bone, and this view has now gained fairly wide acceptance (Jaffe and Lichtenstein 1940, Jackson *et al.* 1949, Golding 1954). As pointed out by Coley (1949) and Willis (1953), however, the status of the lesion is still debatable.

In some respects osteoid osteoma and osteogenic fibroma present a close similarity, but Lichtenstein (1952) and Golding and Sissons (1954) believed that the two processes were different. In two cases classified as osteogenic fibroma by Golding and Sissons (1954), in both of which the lesion occurred in the vertebral column, these authors found that the lesions differed from osteoid osteoma in several respects. They did not cause the severe pain characteristic of osteoid osteoma, and they were considerably larger and showed progressive enlargement with compression of adjacent soft tissues. These features, with the absence of a zone of sclerosis surrounding the lesion, resulted in a totally different radiographic appearance. Pathologically, Golding and Sissons (1954) found in the lesions of osteogenic fibroma the same tissue components as in osteoid osteoma, but the proportions of each and their arrangement were different. In particular, the continuous network of bone and osteoid tissue which make up the " nidus " of osteoid osteoma was absent, and there was much more fibrous tissue. In the case reported here the clinical, radiographic and pathological features resemble those of osteogenic fibroma. Pathologically, the structure of the material was distinctly more irregular and imperfectly differentiated than is usual in the "nidus" of osteoid osteoma, and there was no dense sclerosis of bone adjacent to the tumour-like tissue.

There seems to be a close relationship between osteogenic fibroma of bone and the histologically similar but rare tumours referred to by Lichtenstein (1952) as benign osteoid-tissue-forming tumours of bone. These tumours occur in children and young adults, show a predilection for the bones of the hand and foot, and may become aggressive in the course of their development " and yet fall short of being osteogenic sarcoma."

From our studies of the literature and of the case recorded above, we incline to the view that the lesion described is a benign neoplasm rather than a manifestation of bone dysplasia, and we have accordingly employed the designation osteogenic fibroma of bone. But it is clear that more studies of similar cases are required to establish the true nature of the condition.

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SELECTIVE CONTRACTURE OF THE FLEXOR DIGITORUM SUBLIMIS MUSCLE TO THE RING FINGER

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A twenty-two years old girl complained of progressive inability to extend the ring finger of the right hand for one year. The condition started with pain in the region of the right elbow, extending two or three inches below the medial epicondyle. The pain subsided within a few weeks but the patient developed a flexion contracture of the right ring finger, which could be extended only on full flexion of the wrist. There was no history of injury or of other illness.

On examination, the only remarkable findings were in the right ring finger and forearm. With the wrist in the mid-position the patient could not extend the ring finger beyond 90 degrees at the middle interphalangeal joint (Fig. 1), although the other fingers could be extended fully. Extension of the ring finger by passive movement could be accomplished only when the wrist was completely flexed, and at the same time a tense cord-like elevation was felt in the forearm between the medial epicondyle and the wrist which relaxed completely when the ring finger was allowed to flex.

The position of the distal interphalangeal joint was not altered even when the wrist joint was fully extended, although this movement caused acute flexion of the proximal

FIG. 1
Condition before operation.

interphalangeal joint. There was no radiographic evidence of abnormality in the elbow joint, and routine blood studies and urine examination were normal. A diagnosis was made of chronic myositis with fibrosis of the flexor digitorum sublimis muscle to the ring finger.

Operation—Under local anaesthesia the proximal third of the cord-like structure in the forearm was exposed, this being facilitated by keeping pressure on the finger to produce tension. The cord-like structure was found to be part of the flexor digitorum sublimis muscle; it was severed with the scalpel. The finger could then be extended easily to the same degree

as the other fingers (Fig. 2). Part of the contracted tissue was resected and sent for pathological examination.

Histological examination showed dense collagenous connective tissue with many elongated fibroblast-like nuclei. Only a few striated muscle fibres were seen. Most of this section showed dense collagenous tissue resembling tendon.

Progress—The patient regained full function of the finger. So far there has been no recurrence of deformity and no involvement of other muscles.

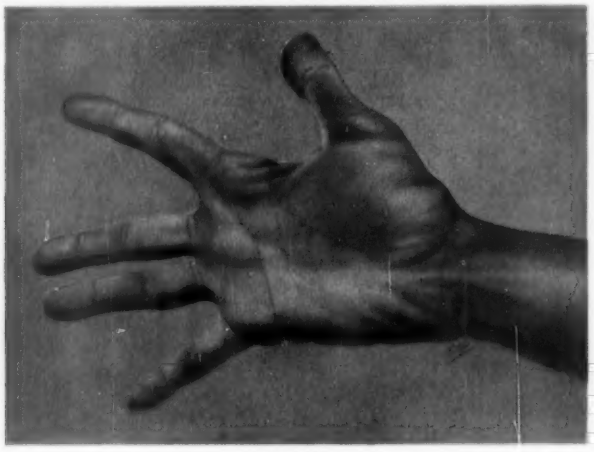

FIG. 2

After division of contracted sublimis muscle.

Comment—The nature of this case is obscure. Localised contracture of one part of the group of forearm muscles has sometimes been observed in Dupuytren's contracture. More probably the changes here represent a fibrositis, and the origin might have been a fasciitis and epicondylitis such as occurs in tennis elbow in the origin of the extensor muscles. The pathology of inflammation of the fascia and of the adjacent origin of the flexor group of muscles of the forearm led to fibrosis of the rest of the muscle fibres to the ring finger. The selectivity of the process to one localised group of fibres is the interesting phenomenon for which reason this case is reported.

RUPTURE OF THE AORTA COMPLICATING TUBERCULOSIS OF THE SPINE

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From Baragwanath Hospital and the South African Institute for Medical Research

In a review of the literature von Zalka (1924) found thirty-three cases of tuberculosis of the aorta, to which he added two of his own. Of these, eighteen were secondary to miliary tuberculosis and seventeen were due to direct spread from surrounding structures—ten from neighbouring lymphatic glands, three from tuberculous caries of the spine, one from a tuberculous pericarditis, one from a lung, one from either lung or spine, and one from an undetermined origin. The demonstration of acid-fast bacilli in the aortic lesion was reported in only three instances of direct extension.

Somerville and Wishart (1948) reported two cases of rupture of the aorta secondary to spinal tuberculosis. In one case it seems likely that the rupture was due to perforation of the aorta by a tuberculous rib. In the second case the final diagnosis was in doubt because the specimen was lost.

CASE REPORT

A Bantu man aged fifty-five was admitted to hospital with a painful swelling on his back, which had been present for two months. It had gradually increased in size. Eight years previously he had sustained an injury to the lumbar spine, for which he had had a spinal fusion.

Fig. 1

Lumbar spine showing an old healed tuberculous lesion of L.2–4, with solid fusion posteriorly.

Examination revealed an oval, tender, fluctuant swelling 7·5 × 10·0 centimetres in size to the left of the mid-line opposite thoracic spines 6 to 12. A lumbar gibbus and a well healed paravertebral incision were also present. There was complete immobility of the lumbar spine and limited mobility of the thoracic spine. On spinal percussion there was no localised tenderness. Further clinical examination revealed no other abnormality. The swelling was diagnosed as a cold abscess and aspirated, 400 millilitres of pus being withdrawn.

Investigations—The pus was sent for bacteriological investigation. Acid-fast bacilli were isolated. Routine Vi, H and O agglutinations were carried out with negative results. The erythrocyte sedimentation rate was 29 millimetres in the first hour. *Radiographs* showed an old healed tuberculous lesion of L.2–4 vertebral bodies, with solid fusion posteriorly (Fig. 1). There was a paravertebral shadow to the left side of the column in the lower thoracic region and a doubtful lesion between the tenth and eleventh thoracic vertebrae (Fig. 2). Penetrating views of the mediastinum, and routine chest views gave no further information, apart from showing pleural thickening on the left side.

Progress—The patient was treated by streptomycin, para-amino-salicylic acid and isonicotinic acid hydrazide without any effect on the temperature, which continued to swing. The abscess had to be aspirated repeatedly. Four weeks after admission the patient complained of severe pain in the epigastrium, aggravated by meals. Three weeks later the patient suddenly collapsed and died.

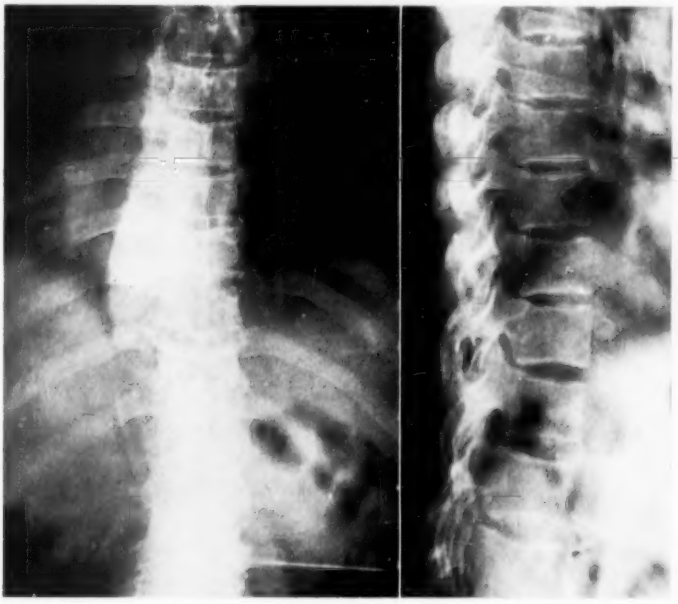

FIG. 2
Thoracic spine showing paravertebral shadow and lesion between T.10 and 11.

FIG. 3
Edge of rupture in aorta. (Haematoxylin and eosin, × 7.)

Post-mortem examination—At necropsy there was a large left-sided paravertebral abscess extending from thoracic vertebrae 6 to 12. The left pleural cavity contained three pints of blood and blood-clot, the lung being collapsed against the mediastinum. There was a circular rupture in the posterior wall of the aorta at the level of the twelfth thoracic vertebra. This rent had smooth rolled edges (Fig. 3), and was in communication with the left pleural cavity

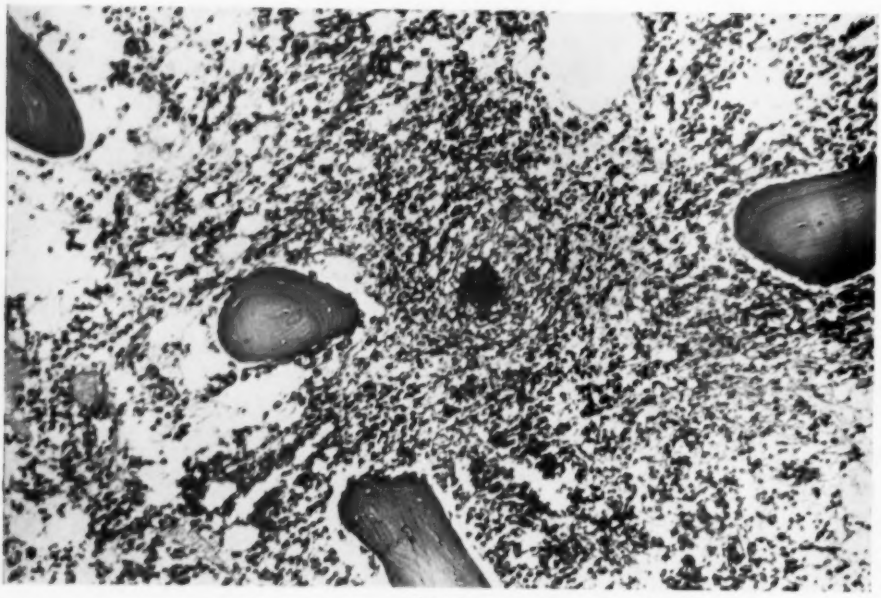

FIG. 4

Tenth thoracic vertebra showing a tuberculous follicle with a central Langhan's type giant cell. (Haematoxylin and eosin, ×120.)

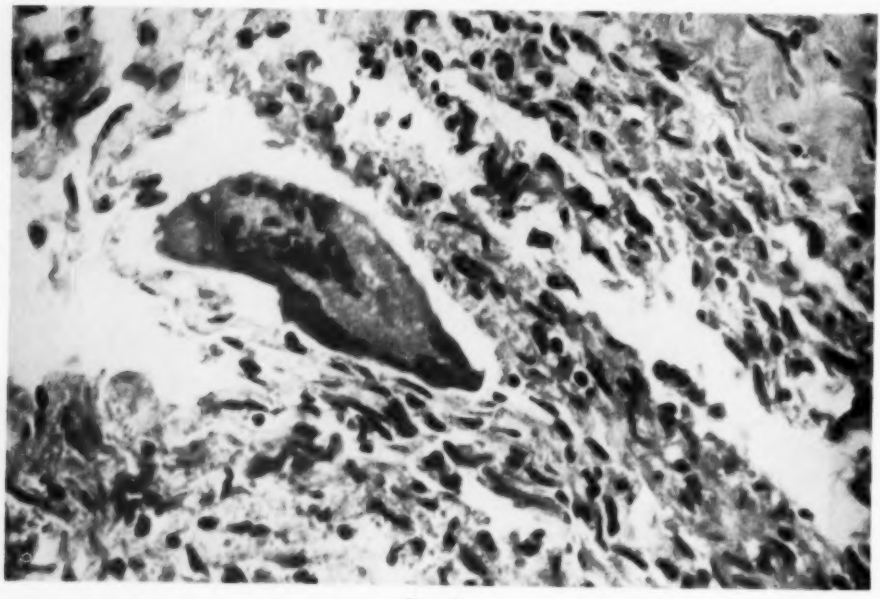

FIG. 5

Wall of the aorta in the region of the rupture, showing a Langhan's type giant cell. (Haematoxylin and eosin, ×480.)

and with a mass of blood clot which had disrupted the deep paravertebral muscles on the left side. The ventral surfaces of the last five thoracic and first lumbar vertebrae were irregular and necrotic. Section of the kyphotic lumbar spine showed it to consist of cancellous bone, with solid fusion of the involved vertebrae.

Histological examination of the wall of the paravertebral abscess, the tenth thoracic vertebra (Fig. 4) and the wall of the aorta in the region of the rupture (Fig. 5) showed the typical features of tuberculosis. Acid-fast bacilli were demonstrated in the sections from the aorta (Fig. 6). Numerous small, translucent white, pin-head nodules were scattered throughout the lungs, kidneys, liver and spleen. On microscopic examination these proved to be miliary tubercles.

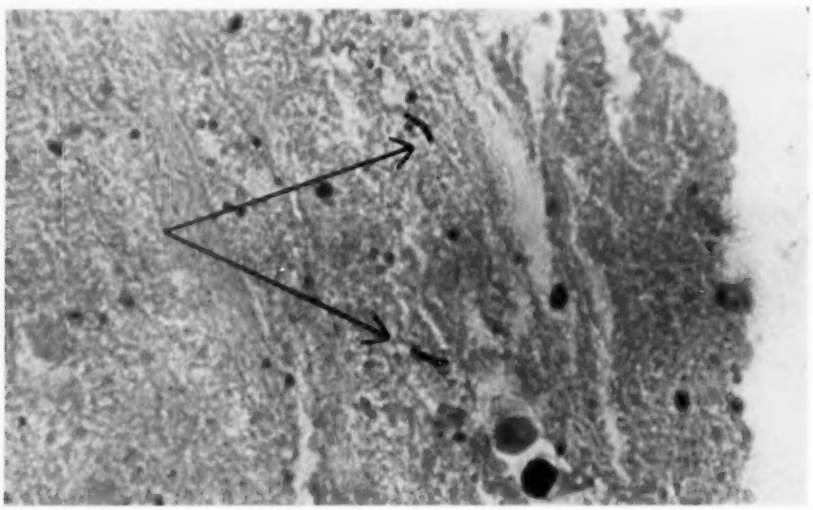

FIG. 6

Wall of aorta in the region of the rupture, showing acid-fast bacilli. (Pottz stain, × 1,200.)

COMMENT

1. The old lumbar lesion appeared inactive radiologically, and this was confirmed at necropsy.
2. It was therefore considered that the paravertebral abscess did not arise from the lumbar spine but was in fact secondary to a new focus of infection in the lower thoracic region.
3. The pathology of the vertebrae was proved on microscopy to be tuberculous.
4. The demonstration of acid-fast bacilli in the wall of the aorta adjacent to the rupture confirmed the existence of a tuberculous aortitis.
5. It seems probable that the rupture into the paravertebral muscles was coincident with the onset of the acute abdominal pain. The sudden death three weeks later was the result of extension and rupture of this false aneurysm into the left pleural cavity.

We are grateful to Dr J. Higginson for his interest and encouragement, and to Dr J. F. Murray for his helpful criticism. The histological sections were prepared by Mrs M. P. Coetzee and the photomicrographs by Mr M. Ulrich, both from the South African Institute for Medical Research.

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FATIGUE FRACTURES OF THE ULNA

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Fatigue fractures are rare in the upper limb. Hartley (1943) stated that fatigue fractures occurred as rarities in the humerus in javelin, discus and grenade throwers, and had been reported in the ulna in diggers. No reference to these can be traced. Two cases of fatigue fracture of the ulna were recorded by Troell, Lauritzen and Möller (1941), and Kitchin (1948) described another case. No other reference to fatigue fracture of the ulna has been discovered in the literature. Two cases are now reported.

CASE REPORTS

Case 1—A right-handed farm labourer, aged thirty and of average intelligence, complained of a painless swelling of the left forearm. One year before, while lifting sacks of oats weighing about one and a half hundredweight, he experienced pain in his left wrist. Ascribing it to a

FIG. 1

FIG. 2

FIG. 3

Case 1. Figure 1—Photograph showing the fusiform swelling of the left ulna. Figure 2—Initial radiograph, one year after the fracture. Non-union is established. Figure 3—Radiograph eighteen months after operation. The fracture is soundly consolidated.

sprain he continued to work. The discomfort lasted only a few days, during which he wore a leather wrist strap. After this he was only aware that the left wrist ached a little when lifting heavy loads, which he continued to do. Three months later he noticed a painless swelling of his left forearm, which he ignored. Six months later, while off work with a cold, he happened to show his doctor his forearm, and thus was referred to hospital. At no time

had he had any pain in the forearm, though he had been doing heavy farm work throughout. There was no relevant past history. His general health had always been excellent, and he had served for five years in the army during the war in category A1.

On examination there was a fusiform, bony hard swelling just proximal to the middle of and continuous with the shaft of the left ulna (Fig. 1). There was no oedema or increase in

FIG. 4

FIG. 5

FIG. 6

Case 2. Figure 4—Initial radiograph. The break in continuity of the cortex can be seen. The fracture line is barely visible. Figure 5—Radiograph nine weeks after fracture. The fracture line is obvious and there is well marked callus formation. Figure 6—Four months after fracture, showing sound union.

temperature at this site, and the skin and underlying muscles moved freely over the swelling. Movements of the left wrist, elbow and radio-ulnar joints were full and painless. There were no enlarged regional lymphatic glands. All other systems were normal. The blood chemistry was normal. The Wassermann and Kahn reactions were negative.

Radiographs showed a pseudarthrosis in the middle of the shaft of the left ulna with much ensheathing new bone (Fig. 2). There was sclerosis in both fragments, especially the upper. Radiographs of the remainder of the skeleton were normal.

At operation the ulnar shaft was exposed and an ununited fracture confirmed. Cortical bone was removed to leave a level platform, and an onlay tibial graft secured in position across the fracture with four screws. The arm was not further immobilised. Histology of the bone removed was normal. Two months after operation he resumed his full farm work. A little initial aching of the wrist soon ceased, and at no time did he have any pain at the fracture site.

One year later he was working full time, with no symptoms except an occasional ache in his wrist. Examination did not reveal any change. All joint movements were full. Radiographs showed the graft well incorporated. There was some further evidence of sclerosis at the fracture site, and as yet little evidence of endosteal repair. He continued at work.

When last examined eighteen months after operation he had no symptoms. Clinically the forearm was unchanged. Radiographs showed the fracture to be soundly consolidated (Fig. 3).

Case 2—A right-handed cook, aged fifty-six, experienced sudden pain in the left forearm while lifting a heavy saucepan. She continued at work. One week later, while cutting up some food, she felt a cracking sensation in the left forearm. The forearm became painful and swollen and she was referred to hospital.

On examination there were tenderness and swelling over the distal third of the left ulna, and pain on movements. Radiographs showed a transverse fissure fracture of the lower third of the left ulna with no displacement (Fig. 4). All other investigations were normal.

An above-elbow plaster was applied. After nine weeks' immobilisation there was still some swelling and a little tenderness at the fracture site. Radiographs showed marked callus formation (Fig. 5). Immobilisation was continued for a total period of three months. When last examined, four months from the date of onset, she had no symptoms, and there were no abnormal physical signs. Radiographs showed the fracture well united (Fig. 6).

REVIEW OF PREVIOUSLY REPORTED CASES

Troell, Lauritzen and Möller (1941) described two cases. The first occurred in a girl aged sixteen while she was lifting a burden of hay on a pitchfork. She experienced a sudden stab of pain in the left forearm. She continued working for eleven days. Later the arm became swollen and tender. Radiographs three weeks later revealed a fracture of the left ulnar diaphysis " in the way of recovery, with pronounced callus formation." This was reported healed in five months.

The second case occurred in a girl, aged sixteen, under similar circumstances, while she was pitching hay into a barn loft. She experienced sudden pain in the right forearm. The arm was swollen and ached the same evening. She continued working for sixteen days. Radiographs showed a fracture of the right ulnar diaphysis " with excess callus formation." This was reported healed in eight weeks.

In neither case was the exact site of fracture or the method of treatment recorded.

Kitchin's (1948) case occurred in the middle of the shaft of the left ulna, in a healthy right-handed farm labourer aged twenty. Pain in the forearm had started three weeks before he was seen, while he was carting forkloads of stable manure. The pain did not prevent him from continuing at work. Examination revealed a fusiform swelling of the middle third of the left ulna, with heat, tenderness and slight oedema. There was a full range of elbow movement. Radiographs showed a fracture of the midshaft of the ulna, with considerable callus and a periosteal reaction extending upwards and downwards along the greater part of the diaphysis. He was treated in a plaster for ten weeks and resumed light work at four months. The fracture was soundly consolidated at fourteen months.

DISCUSSION

The similarity between Case 1 and the previously reported cases is striking. Each fracture occurred in a young healthy person engaged in farm work. In three cases this entailed lifting heavy loads of manure or hay on a fork, and in the fourth lifting heavy sacks of oats. In three cases, though pain was present at the fracture site, the patients were able to continue working for periods up to three weeks, and in Case 1 pain was so conspicuously absent that it was nine months before the patient sought medical advice.

In two cases the fracture occurred in the left ulna of right-handed men. In the other two cases, one occurred in the left and one in the right ulna, no mention being made of the master hand. In Kitchin's case " the left forearm supported both the downward thrust of the right hand and the pull of the resisting load," and in my Case 1 both forearms supported the weight of a considerable load. In each case the position of the forearm at the time of

strain was that of full supination with the elbow flexed to about a right angle. Thus in each case the weight was taken approximately at right angles to the ulnar shaft.

From the similarity of the history and clinical findings it seems probable that the mechanism of injury in Troell, Lauritzen and Möller's cases was the same. If so, then one must assume that in the case with the right ulnar fracture, the patient was left handed. Unfortunately this was not recorded.

In my Case 2 the fracture occurred in an older patient. The position of the upper limb at the time of fracture is not known. It seems possible that in lifting a heavy saucepan the forearm would be similarly supinated and the elbow flexed.

The site of fracture in Kitchin's case and in Case 1 was just proximal to the middle of the shaft of the ulna and about one inch distal to the usual site of entry of the nutrient artery. At this point the ulna shaft is at its widest because the interosseous ridge is well marked. In Case 2 the fracture occurred in the lower third of the ulna at a site where the shaft is at its narrowest.

At both these sites there is a well marked change in cross-sectional shape of the ulnar shaft (Fig.7). This may be a potential source of weakness. Nevertheless there does not appear to be any significant alteration in the ratio of compact to cancellous bone at these sites, as measured on serial sections.

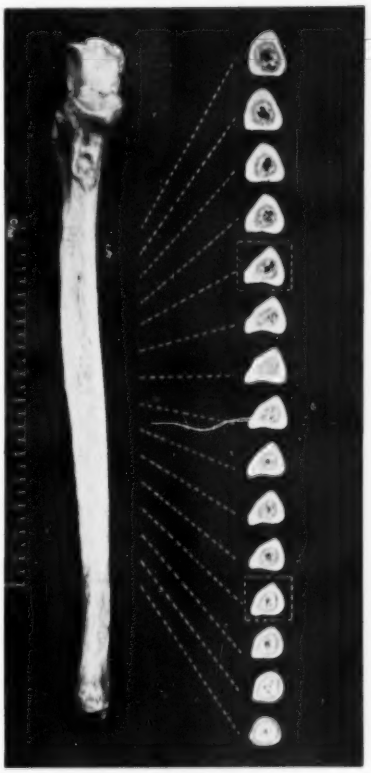

FIG. 7

Photograph of an adult ulna and corresponding serial sections at intervals of one centimetre. The sites of fracture in Cases 1 and 2 are shown.

The fatigue fracture of the metatarsal is commonly called a " march " fracture. Burrows (1948) labelled the low fibular fracture a " running " fracture and the high fibular fracture a " jump " fracture. The ulnar fatigue fracture appears to be a " lifting " fracture.

SUMMARY

1. Two cases of fatigue fracture of the ulna are reported.
2. Three cases previously reported are reviewed and a striking similarity with one of the present cases is noted.
3. In all cases the mechanism of injury appears to have been the same.

I wish to record my thanks to Mr E. P. Brockman and Mr C. W. Fleming for permission to report their cases, and to the Photographic Departments of Westminster Hospital and the Institute of Orthopaedics, Royal National Orthopaedic Hospital, for the illustrations.

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OS ODONTOIDEUM

A Congenital Abnormality of the Axis: Case Report

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Congenital abnormalities of the axis have long been known but are not often described. Their frequency is unknown, for unless symptoms demand a radiographic examination of the cervical spine they pass unnoticed. In the case described, symptoms led to radiographic examination of the neck, but it seems likely that the appearances discovered were irrelevant to the symptoms and signs.

The odontoid process at birth is separated from the body of the axis by cartilage. The process begins to fuse with the body in the fourth year of life, but some cartilage remains at the centre of the process until an advanced age. Sometimes the odontoid process fails to develop at all; this anomaly has been described by Roberts (1933), Weiler (1942), Scannell (1945) and Stiefel (1950), both with and without symptoms of atlanto-axial subluxation. The process may be ossified but detached, forming an os odontoideum. This anomaly has been rarely described. It is mentioned in older anatomy text-books, and Nievergelt (1948) described a number of examples and gave references to more. The case here reported seems to be another.

FIG. 1
Plain radiographs of cervical spine.

CASE REPORT

A man aged fifty-five fell down at home "in a faint." He could not remember the episode clearly. He recovered quickly and came to hospital. He vomited soon after arrival. He gave a history of a somewhat similar attack of "fainting" some years before. He was admitted under a physician. Complete clinical examination revealed no abnormality except some bruising of the back of his head but there were no signs suggestive of bone damage or of injury to the brain or spinal cord. He was discharged five days later.

He did not attend hospital again for four months, when he complained of general symptoms of fatigue and some pain in the shoulders. It was noted that he had some pain on neck flexion, but no other signs were observed. Radiographs of the cervical spine (Fig. 1) appeared to show a subluxation of the atlanto-axial articulation. He was referred for an orthopaedic opinion.

FIG. 2
Antero-posterior and lateral tomographs of cervical spine.

Clinical examination of the neck showed no abnormality. He could move his head freely and painlessly in all directions. No abnormalities were found in the nervous system. The symptoms in the neck were confined to the lower cervical and shoulder regions and could not in any way be related to disturbance in the upper part of the spinal cord or to the upper cervical roots.

Tomography was subsequently carried out and showed an entirely separate odontoid process, displaced upwards and forwards from the body of the axis, surrounded by what appeared to be a smooth cortex (Fig. 2).

COMMENT

It is hard to believe that the separation of the odontoid process was due to a fracture four months old, especially as at no time were there signs to suggest a serious cervical bone lesion. It may be that the relative instability of the atlanto-axial joint was responsible either for the "fainting" episode, or for the slight neck symptoms after the fall, or for both. That is a matter for speculation: what seems more certain is that this is an example of an os odontoideum. On that assumption, no treatment was advised.

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MULTIPLE PSEUDO-CYSTIC TUBERCULOSIS OF BONE

Report of a Case

C. L. Clinton-Thomas and W. B. Young, Malaya

Multiple pseudo-cyst formation is an uncommon manifestation of skeletal tuberculosis. Confusion has been caused by the failure to differentiate between this form of osseous tuberculosis, the solitary foci of diaphysial tuberculous osteitis, tuberculous dactylitis, and the bone lesions of sarcoidosis.

This confusion has largely been due to Jüngling (1920, 1928) who, believing them to be of tuberculous etiology, gave the name osteitis tuberculosa multiplex cystica (afterwards changed to cystoides to indicate their pseudo-cystic nature) to the lesions he found in the small bones of the hands and feet in certain cases of sarcoidosis. Since then cases of tuberculous cystic bone disease with lesions quite unlike those associated with sarcoidosis have been given the eponymous title or described as examples of osteitis tuberculosa multiplex cystica or cystoides.

This anomaly has not passed unnoticed, for Ellis (1940), Sweet and Abramson (1941), Golding (1950), Alexander and Mansuy (1950), and Komins (1952), among others, have drawn attention to it. That confusion still exists, however, is shown in a recent paper by Girdwood (1953) in which an atypical case of tuberculous dactylitis was described as an example of Jüngling's disease.

Komins (1952) has attempted to clear up the muddle in the early literature. He accepted thirty-one cases of multiple cystic tuberculosis of bone in childhood from the English literature, reported three more of his own, and suggested that the bone lesions of sarcoidosis should be called osteitis multiplex sarcoidosa.

Additional cases have since been reported by De Pape (1954) and by Murray (1954). Murray described the characteristic features, of which the most important were: the predilection for childhood; the tendency for the lesions to affect the long bones symmetrically and simultaneously and to progress and regress in harmony; the rarity of joint involvement and the favourable prognosis if it did occur; the lack of early bone reaction with later cortical expansion and fusiform enlargement; and the frequent occurrence of sinuses without sequestrum formation.

The case to be reported agrees with these criteria and demonstrates the effect of streptomycin on this type of bone tuberculosis.

CASE REPORT

A Chinese boy aged three years was admitted to hospital with a four months' history of discharging sinuses in the right orbit, the left foot and both forearms. All the sinuses had been preceded by painless swellings which had either broken down spontaneously or had failed to heal after incision. He was the only child of intelligent healthy parents of good social standing. He had not suffered from any serious illness before, and there was no history of contact with tuberculosis.

Clinical examination—He was a healthy looking child, normal in size for his age. The temperature and pulse were normal. There were painless, fusiform bony swellings of the distal ends of both forearms, with two sinuses on the left wrist and one on the right. There was a swelling of the distal part of the left foot with two sinuses. There was a sinus, involving the upper eyelid, at the outer canthus of the right eye. On examination of the chest there was diminution of air entry and dullness on percussion at the right apex.

Radiological examination—A radiograph of the chest (Fig. 1) showed collapse and consolidation of the right upper lobe, with enlargement of the hilar lymph nodes; the appearances were typical of a primary tuberculous complex. There was a destructive lesion of oval shape in the posterior part of the right sixth rib. The radiographs of the forearms demonstrated multiple cyst-like lesions. The areas of destruction were oval, with regular, clearly defined margins. There was cortical expansion and in some areas fusiform enlargement of the bone with a little subperiosteal new bone formation (Figs. 2 and 3).

Radiological examination of the remainder of the skeleton revealed similar lesions in many of the bones (Figs. 4 to 12).

A diagrammatic representation of the radiological findings (Fig. 13) indicates the tendency for the lesions to be symmetrically and centrifugally situated with reference to the axial skeleton and for the distal parts of the limbs to be affected.

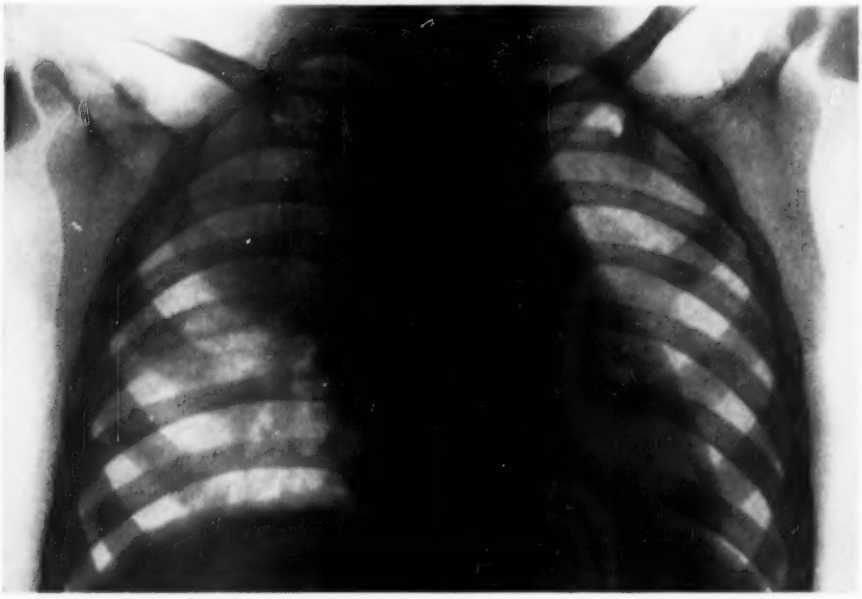

FIG. 1
Radiograph of chest on admission.

Investigations—The Mantoux test was strongly positive with 0·1 millilitre of 1/1,000 old tuberculin. The Kahn test was negative. The serum calcium was 9·5 milligrams per cent, and serum protein 7·3 grammes per cent. The albumen-globulin ratio was normal. The erythrocyte sedimentation rate was 20 millimetres in the first hour (Westergren).

Sputum was not available, but repeated examinations of gastric washings and faeces were negative for acid-fast bacilli. Curettage of the sinuses and culture of the scrapings, on two occasions, gave a poor growth of staphylococcus aureus. No fungus was grown, and direct smear and culture were negative for acid-fast bacilli.

Biopsy from right ulna—The naked eye appearance was that of a soft granuloma which shelled out of the bone with ease, leaving a smooth cavity. There was no pus or caseous material. Culture of the specimen yielded no acid-fast bacilli or other organisms.

Histologically the specimen consisted of inflammatory granulation tissue replacing bone and bone marrow. Numerous multinucleated giant cells were present and the inflammatory tissue was studded with a number of characteristic " tubercles " each consisting of giant cells,

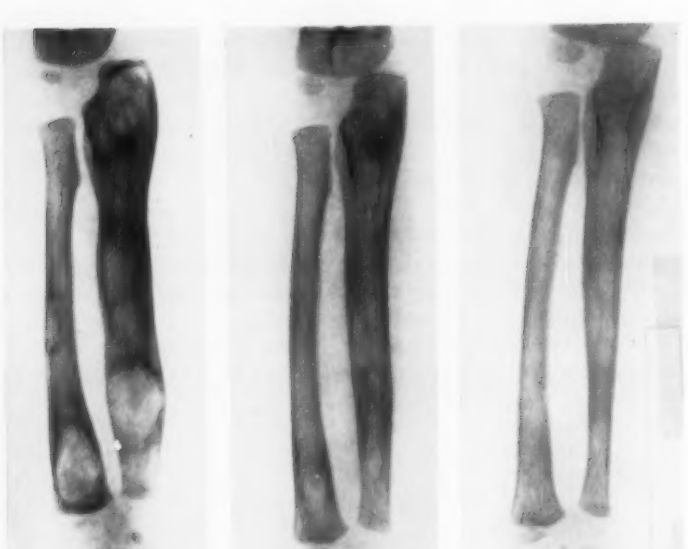

FIG. 2 FIG. 3 FIG. 4

Right radius and ulna. Figure 2—On admission. Figure 3—After three months. Figure 4—After ten months.

FIG. 5 FIG. 6 FIG. 7

Further radiographs at time of admission. Figure 5—Right humerus. Figure 6—Left humerus. Figure 7—Left radius and ulna.

FIG. 8

FIG. 9

FIG. 10

Lower limb bones at time of admission. Figure 8—Part of left femur. Figure 9—Left tibia and fibula. Figure 10—Right tibia and fibula.

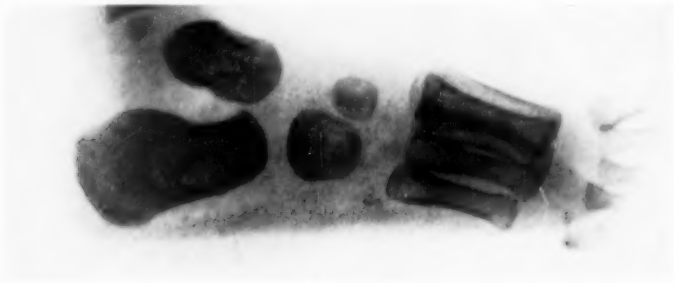

FIG. 11

FIG. 12

Right foot (Fig. 11) and left foot (Fig. 12) at time of admission.

epithelioid cells and fibroblasts. Occasionally areas of caseation were present in the inflammatory tissue (Figs. 14 and 15). Although acid-fast bacilli were not demonstrated in the section the histological evidence of tuberculosis was very strong (Dr H. A. Sissons).

Treatment and subsequent progress—Streptomycin (grammes ½ daily) and para-amino-salicylic acid (grammes 6 daily) were given for 100 days. The forearms and left lower leg were immobilised in light plaster splints. The child never looked ill and at no time had a cough. His appetite was good and he gained weight satisfactorily. The temperature and pulse remained normal. The erythrocyte sedimentation rate fell to 10 millimetres/hour two weeks after treatment had begun and thereafter remained below 5 millimetres/hour.

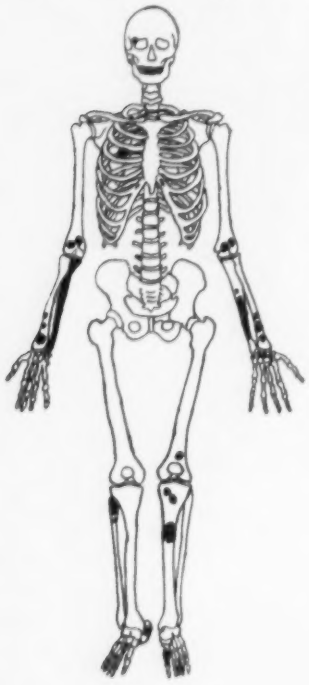

FIG. 13
Diagram showing the distribution of the lesions.

Three weeks after treatment by streptomycin was begun the sinuses began to heal; that in the orbit was healed at one month and the remainder were healed in two months.

Radiological examination of the skeleton three months after admission and shortly before the child's discharge from hospital gave evidence that a considerable degree of healing had already taken place.

A further radiological examination seven months later showed that all the affected bones had regained their normal shape and that bone texture in most sites had resumed its normal appearance.

Radiographs of the right forearm taken at these times illustrate the process of healing (Figs. 3 and 4).

The child's general condition remains good, but a recent radiograph of the lungs still shows partial atelectasis of the right upper lobe.

DISCUSSION

Despite the failure to isolate mycobacterium tuberculosis from the sputum, stomach, faeces or one of the bony lesions, the radiographic appearance of the lungs, the positive Mantoux reaction, the histological findings and the rapid disappearance of the bony lesions under streptomycin therapy are almost conclusive evidence of a tuberculous etiology in this case.

Most of the recorded cases of multiple pseudo-cystic tuberculosis of bone affecting children (Table I) have occurred in under-nourished coloured infants in tropical or semi-tropical countries, but in our experience the usual case of skeletal tuberculosis in these regions in no way differs from that found in more temperate and socially advanced parts of the world, and the child in this case showed no evidence of malnutrition.

Although analysis of the recorded cases shows that a number of children died of generalised tuberculosis the relatively benign character of multiple pseudo-cystic tuberculosis of bone, the tendency to spontaneous regression and the rarity of joint involvement with a favourable prognosis if it does occur, have been noticed by several observers. It is indeed a striking feature that such widely disseminated disease may cause so little constitutional disturbance and heal leaving so little evidence.

In pseudo-cystic tuberculosis of the bone the widespread distribution of the lesions, their simultaneous onset and the similarity of their radiographic appearances are evidence of haematogenous dissemination over a short time.

It is tempting to suppose that the organism in these cases might have a lowered virulence

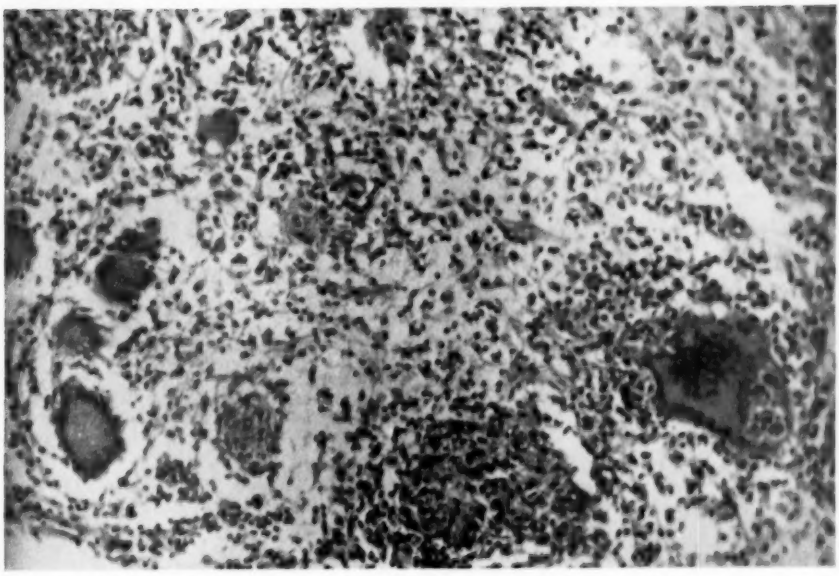

FIG. 14

Photomicrograph showing granulomatous tissue with scattered multinucleated giant cells. (×108)

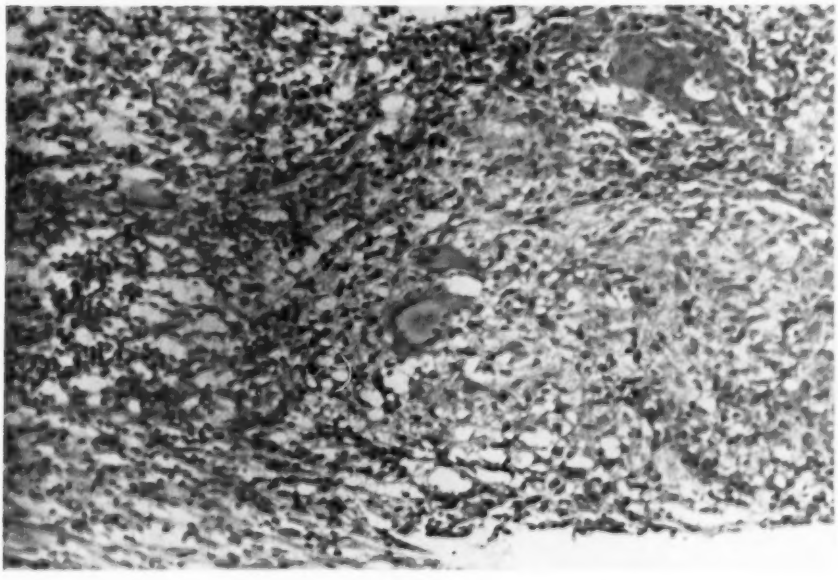

FIG. 15

Bone marrow, showing a tubercle consisting of two multinucleated giant cells surrounded by epithelioid cells and fibroblasts. (×200)

TABLE I

RECORDED CASES OF MULTIPLE PSEUDO-CYSTIC TUBERCULOSIS OF BONE

Author	Race	Age	Proof
Lovett and Wolbach (1920)	Not stated	20 months	Biopsy
Lovett and Wolbach (1920)	Not stated	2¼ years	Biopsy
Schwentker (1931)	European	1 year	Animal inoculation + ve
Vastine and Bacon (1935)	Negro	17 months	Biopsy
Vastine and Bacon (1935)	Negro	2 years	Mantoux + ve
Thornton (1938)	Negro	13 years	Biopsy
Law and Perham (1938)	European	6 years	Mantoux + ve
Connolly (1938)	European	2½ years	Biopsy
Martin (1939)	Negro	6 years	Biopsy
Hodges (1939)	Negro	13 years	Autopsy
Hodges (1939)	Negro	9 months	Biopsy
Carrell and Childress (1940)	European	7 years	Biopsy
Martin and Fogel (1941)	Negro	1½ years	Gastric wash T.B. + ve
Sweet and Abramson (1941)	Negro	3 years	Biopsy
Ziady and Selzer (1942)	Negro	7 months	Biopsy
Frost (1942)	Negro	18 months	Autopsy
Monfort and Solomon (1942)	Negro	16 months	Biopsy
Koo and Yang (1946)	Chinese	11 months	Tuberculous gland
Birdsong and L'Engle (1948)	European	13 months	Mantoux + ve
Kreidberg and Downing (1948)	European	11½ years	Biopsy
Nagah (1948)	Egyptian	1½ years	Biopsy
Turkish *et al.* (1949)	Negro	19 months	Biopsy
Kutz *et al.* (1950)	Negro	11 months	Mantoux + ve
Kutz *et al.* (1950)	Negro	21 months	Mantoux + ve
Komins (1952)	Bantu	1 year	Mantoux + ve
Komins (1952)	Bantu	1¾ years	Biopsy
Komins (1952)	Bantu	7 years	Gland biopsy
Murray (1954)	European	4 years	Animal inoculation + ve
De Pape (1954)	Red Indian	6½ months	Pus T.B. + ve
Clinton-Thomas and Young (1955)	Chinese	3 years	Biopsy

or be of a different type, but in cases in which there has been bacteriological proof the organism has not shown cultural or morphological differences and animal inoculation has not demonstrated any altered virulence.

Any explanation of this uncommon form of skeletal tuberculosis must inevitably be a pure hypothesis, but we believe that a possible solution may be found in a study of the time relationship of the initial bacillaemia to the primary lung lesion. It is difficult to prove in man that bacillaemia is a regular feature of a primary infection, but it is a constant finding in animal experiments (Wilson 1933), and in all reported necropsies in early cases of primary complex tubercles have been found widely distributed throughout the body.

According to Wallgren (1948) there is an interval of five or six weeks between the primary infection and the development of hypersensitivity to tuberculin. The usual lesion of bone and joint tuberculosis occurs after the establishment of an allergic state.

If on rare occasions the bacillaemia should occur before the development of tuberculin sensitivity and bacilli were able to gain a foothold in the bones with sparing of other more vital organs, it is reasonable to suppose that the response might be quite different from that found after allergy had developed, and that the subsequent pattern and progress of the disease might be substantially altered.

SUMMARY

1. A case of multiple pseudo-cystic tuberculosis of bone presenting as a generalised disease of the skeleton in a child is reported.
2. The response to streptomycin and para-amino-salicylic acid is shown.
3. A possible explanation of the etiology of this rare disease is suggested.

We wish to express our thanks to Dr Campbell Golding of the Royal National Orthopaedic Hospital, London, for his help and advice, and to Dr H. A. Sissons, of the same hospital, for the histological report and the photomicrographs.

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ORTHOPAEDIC PROBLEMS OF HEREDITARY SENSORY NEUROPATHY

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Petrie (1953) described a patient with multiple arthropathy from congenital indifference to pain. Cram (1953) reviewed neuropathic derangement of joints associated with diabetes mellitus. We have studied the family reported by Heller and Robb (1955), who traced the disorder through six generations, with evidence of hereditary sensory neuropathy in five patients. Three other members of the family showed an incomplete form of the disease, one also having an affection of the spinal cord, probably from multiple sclerosis. Price (1913), Schultze (1917), Hicks (1922), Ogryzlo (1946) and Denny-Brown (1951) reported cases. Denny-Brown showed histologically that the fundamental pathology was in the dorsal root ganglia. There was demyelinisation of the afferent fibres and a paucity of cells in the ganglia. Around these, a satellite proliferation of Schwann cells could be noted, small nodules being formed. Within these cellular nodules were dendritic proliferations. This was described as the essential pathology in hereditary sensory neuropathy.

Each of the following cases shows 1) severe trophic disorders of all extremities; 2) loss of all sensibility in the extremities, in a glove-and-stocking distribution; and 3) loss of deep tendon reflexes. The peripheral pulses, sweating, temperature and other manifestations of sympathetic function were normal.

CASE REPORTS

Case 1—A man of twenty recalled that when he was about five years of age he had difficulty, in walking, from tripping over small objects. He also had difficulty in buttoning his shirt because of a lack of feeling in his fingers. At the age of nine he scratched the toes of both feet while swimming, and the scratches took a long time to heal. Later the same year he experienced a dull burning sensation in his feet, unrelated to activity. An operation of unknown nature was performed on both feet, and he was in plaster for six months. Thereafter he enjoyed good health until he reached the age of twelve, when he noticed that his nails began to crack, particularly on the second and third right fingers. Pus accumulated painlessly under the nail, and the condition progressed until the nails and a small piece of bone were removed from the second and third phalanges. The wounds healed well.

In that same year he suffered recurrent infection of the dorsum of the left foot. Later he underwent incision, drainage and sequestrectomy of the lower third of the left tibia. Radiographs at that time showed marked degenerative changes of the foot. The onset of the various febrile episodes is of interest. He first noticed a cough, with a feeling of discomfort over the lower retrosternal area. This coughing would be followed by malaise, sweating, chills and fever. His oral temperature would reach 103–104 degrees Fahrenheit. So long as the pyrexia continued, the cough persisted. This would last for several days, the patient remaining in bed. Lysis of the temperature was associated with a feeling of well-being. No marked pain other than a dull widespread ache would occur in the extremity, but an area of infection was always found on examination.

At the age of fifteen years the tibial osteomyelitis had progressed, and a below-knee amputation was carried out. The stump healed slowly, and when he began to use a prosthesis the stump became ulcerated. The records of the following years reveal ten admissions to hospital for various procedures for the infected stump and for osteomyelitis of the right foot.

For the past two years there has been a persistent sinus in the foot, necessitating excision of the third and fourth metatarsals. The fingers have become progressively more ulcerated and several sequestra have been discharged. At present there is a large ulcer on the sole of the foot.

The whole life of this patient has been governed by these recurrent episodes of infection. As each occurs, he remains at home and elevates the part until healing occurs.

On examination the head, thorax, abdomen, shoulders, elbows and hips were normal. A photograph of the patient is shown in Figure 1. The hands showed thickened skin over the palmar and dorsal surfaces of the fingers (Fig. 2), the distal phalanges of which were missing. Ulcers were scattered throughout the fingers. The skin of the left popliteal fossa was thickened and the whole stump was irregular, the skin being of variable roughness. There was a discharging sinus at the anterior surface of the stump. The right foot was in slight valgus; there was only a small range of movement at the ankle and subtalar joints. The dorsum of the foot was reddened, indurated and warm and showed the scars of previous sinuses and operations. The sole of the foot was ulcerated (Fig. 3).

FIG. 1

Case 1—Photograph of the patient, showing lesions on fingers and deformity of right foot and ankle.

Neurological examination showed normal cranial nerves, muscle groups and co-ordination. All superficial and deep reflexes were absent. The sense of touch and pain was absent below the knee and over the forearms and hands, with hypaesthesia fading to the mid-thigh level and above the elbow. Temperature sensation was absent below the lower forearms and below the knees. Position sense was absent in the toes of the right foot and diminished in both

FIG. 2

FIG. 3

Case 1. Figure 2—Right hand showing shortened fingers, ulceration, and thickened creases. Figure 3—Right foot, showing a large ulcer on the sole.

little fingers. Vibration sense was lost over the fingers of both hands, right toes and left stump, and diminished over the right tibial surface. All areas of sensory loss had fading borders. *Radiographic examination*—The right ankle showed narrowing of the joint space with irregularity of the anterior part of the articular surface of the tibia. Hypertrophic changes

FIG. 4

Case 1—Radiograph of right foot. The talus shows sclerotic degenerative changes with narrowing of its articular margins.

were shown at the neck of the talus (Fig. 4). The subtalar joint was almost obliterated. The talo-navicular, navicular-cuneiform, and first and second metatarso-cuneiform joints all showed a similar change. The bones were sclerotic and irregular. The right third and fourth

FIG. 5

FIG. 6

Case 1. Figure 5—Right foot, showing destruction of the third and fourth metatarso-phalangeal joints. Figure 6—Right hand showing loss of phalanges.

metatarso-phalangeal joints were destroyed and the second distal phalanx was represented by only a small fragment of bone (Fig. 5).

The hand showed narrowing of the right first metacarpo-phalangeal joint with medial and lateral osteophyte formation of the proximal phalanx (Fig. 6). The distal phalanx of the thumb was fragmented, as were those of the right and left little fingers and the left fourth

finger. The middle phalanx showed marked loss of substance in the second and third fingers and in the fourth finger of the right hand.

Case 2—This boy, the brother of the patient described in Case 1, was eighteen years old at the time of examination. His history is similar to that of his brother, progressive degenerative processes taking place at the periphery of the limbs. When he was four the mother noticed fissuring of the toe-nails, which eventually separated. At the age of nine he suffered a burn to his right hand when touching a stove. This did not cause any pain. Subsequent skin necrosis and spontaneous extrusion of bony fragments occurred from the finger tips, the wounds healing slowly. Similar incidents have recurred over the years, the most recent being three months ago. At that time a painless inflammation of the right third distal phalanx of the hand occurred, resulting in a loss of that part.

FIG. 7

Case 2—Right and left feet. The left shows marked swelling and deformity. Note trophic changes of the nails.

When he was twelve he fell and fractured the right femoral shaft. The fracture was treated by open reduction and internal fixation, but despite prolonged immobilisation in plaster union was delayed. After a further operation the femur became infected. When he was seventeen he fractured his left femoral shaft in another fall. This fracture was treated by skeletal traction and later by immobilisation in plaster.

While he was walking with crutches he developed a swelling on the left foot, with a dull ache, and this degenerative process has progressed. He also developed an infected ulcer at the right fifth metatarsal, which was later excised. At age eighteen an inflammatory process occurred at the lateral malleolus of the left ankle. After surgical drainage this wound eventually healed. This patient, like his brother, suffered malaise and fever before he would notice any inflammation of a part.

On examination, both hands showed loss of the distal phalanges. The skin was thickened, with small dry ulcerations at several sites.

There was an inch of shortening of the left lower limb. The right foot was in slight

valgus (Fig. 7), and the range of ankle and subtalar movement was impaired. The fifth metatarsal and phalanges were absent and the other toes were deformed. The left foot and ankle were markedly deformed (Fig. 7). The range of ankle movement was only 20 degrees, and of tarsal movement nil. Both malleolar regions were markedly swollen and indurated and there was a sinus over the lateral malleolus.

Neurological examination revealed normal cranial nerves, musculature and co-ordination. All abdominal and deep reflexes were absent. The plantar responses were normal. Peripheral sensory loss was complete over the hands and feet, and faded towards the shoulders and waist. *Radiographic examination*—There was slight spondylolisthesis at L.5–S.1. The uppermost third of the right femur was irregular and sclerotic—the site of previous fractures. The

FIG. 8

FIG. 9

Case 2—Radiographs of feet. On the left side (Fig. 9) the talus and the adjacent joints show marked degenerative changes.

right ankle showed only slight changes (Fig. 8), but the left ankle and foot showed severe destruction that has progressed rapidly in the past two years (Fig. 9). The ankle joint was greatly narrowed. The changes were confined to the calcaneum, talus and navicular, with their adjacent joints. Marked sclerotic changes have occurred of each of these bones with fragmentation of the navicular. The distal phalanx of the fourth toe was partly destroyed. The hands showed changes like those in Case 1.

Case 3—This thirteen year old girl is the youngest sibling. When she was six she injured her right foot and suffered some loss of skin. Three weeks later she noticed a vague dull ache over the whole of the foot, which was enlarged but not reddened. After rest in plaster the inflammation subsided but the foot was never restored to normal. By the age of ten the right foot had developed a painless inflammation of the fifth toe. She remained in bed for three months, but the ulceration progressed. Later she fractured the left femur in a fall. When she was twelve the right fifth toe ulceration had progressed to the degree that osteomyelitis of the metatarsal had occurred. The distal part of the metatarsal and the phalanx were excised. A biopsy of the right lateral femoral cutaneous nerve was obtained at that time. The pathology was as reported by Denny-Brown (1951). At about that time successive fractures of various metatarsal bones occurred. Some were related to injury, whereas others were attributed to stress phenomena. Union occurred in all cases.

On examination, the present time, the patient was able to walk only with crutches. A small ulcer was present over the lateral aspect of the right cuboid bone. Part of the

right fifth metatarsal and all of the phalanges were absent (Fig. 10). An area of redness was present at the lateral aspect of the base of the left fifth toe, and several of the toes were deformed. The right index finger showed inflammatory changes of the distal phalanx with some loss of substance at the tip. Trophic nail changes were present.

Neurological examination revealed normal cranial nerves, musculature and co-ordination. All abdominal and deep reflexes were absent except the triceps jerk, which was sluggish on both sides. There was no Babinski sign. Sensation to touch and to pain was absent over the periphery of all four limbs, with fading borders towards the shoulders and thighs. Temperature sensation was present to a slight degree over the toes and fingers, but vibration sense was absent in these areas. Impaired appreciation of position sense was noted in the fingers and toes.

FIG. 10

Case 3—Right foot showing ulceration over the cuboid area. The skin shows patches of vitiligo.

Radiographic examination—The right foot showed osteolytic destruction of the fifth metatarsophalangeal joint, the site of the skin ulcer. The distal phalanges of the fourth fingers and of the left fifth finger showed early irregularity at their extremities. There was slight spondylolisthesis at L.5–S.1.

DISCUSSION

The patients described all began to have symptoms in the first decade of life. The initial and some subsequent problems have had their origin in the damage done to the skin. Others have been due to progressive joint destruction. In spite of having other members of the family suffering from this disease, the youngest sibling could not limit her activities in order to prevent a similar sequence of events for herself. Her problem at the present time is ulceration of the lateral aspect of the right tarsus. Normal care of the feet and the use of proper shoes have not been sufficient to prevent the skin necrosis. It is probable that she too will suffer destructive changes of the foot and ankle, with perforating ulcers.

The pathological processes at work in the various joints are similar to those described

in tabes dorsalis and named Charcot joints. The constant abuse of the bony structure, its movements unlimited by reflex or pain activity, allows for the development of traumatic arthritis. This progresses, fragments of bone being released and joint spaces being obliterated. This is shown well in the radiographs of the feet. In Case 2 the patient's left foot is unstable, and it is not controlled well by a surgical boot and side-bar. Though arthrodesis might be attempted it seems likely that amputation will be required. But below-knee amputation through an area of hypaesthesia has its own problems, as illustrated in Case 1. The skin is of poor texture and a draining sinus persists. This area still has a moderate sensory deficit. Supracondylar amputation may have been a better choice. With the frequent occurrence of infected ulcers of the right foot, the outlook is most gloomy.

Though the cases of Tocantins and Reimann (1939) may not have been of the same etiology, the management of their problem was similar. They described a man of fifty-three who was able to continue at work despite multiple amputations. The family reported by Heller and Robb (1955) also included a man of eighty-two. He has lived his life without major surgery being performed, though he suffered a spontaneous right mid-tarsal amputation. Several small ulcers exist at his stump. One should not by-pass the best and obvious method of therapy, distasteful as it may be. Suffice to say, there is room for conservative treatment and it should be used to the full.

The management of these perforating ulcers is based upon complete rest, constant attention to the wounds, and antibiotics. Most of the problems have been of the lower extremity. Weight bearing has had to be either reduced or eliminated. Though bed rest is the desirable objective, most of these people do not have the patience to remain recumbent for many months several times each year. Crutches may be used, alone or in combination with plaster-of-Paris.

When ulcerations constantly occur over a bony prominence the question of resection of that area should be considered. However, a word of caution: not infrequently, the soft tissues are the site of a chronic inflammatory process. This leads to slow healing of the wound, as noted in Case 1.

SUMMARY

1. Three cases of hereditary sensory neuropathy are reported.

2. Neuropathic destruction of joints and chronic infected ulcers are the primary problems of management.

3. This entity must be considered in the differential diagnosis of trophic ulcerations of the extremities.

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EXPERIENCES WITH EXPERIMENTAL PLASTER BANDAGES CONTAINING RESIN

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For some time now in this country various orthopaedic centres have been using plaster-of-Paris bandages dipped into water containing melamine formaldehyde resin and catalyst, claiming a stronger plaster, lighter in weight but more durable although requiring less material. These plasters also have the advantage of being more resistant to water and soiling with urine. A drawback is the necessity for preparing two special solutions which have to be mixed immediately before use.

Recently we have been given a trial supply of proprietary British-made plaster bandages containing the resin incorporated in the bandage.*

RESULTS

In two months' experience with these plasters we have noted the following advantages.
1) A saving of about 33 per cent of material will give a plaster of the same strength, which is lighter and yet more durable, withstanding exposure to moisture better.
2) There is less mess when the plaster is applied, because the plaster cream is more cohesive.
3) The bandage can be squeezed hard without loss of plaster material.

The disadvantages are: 1) The soaking time is slightly longer; and 2) the dried plaster is more difficult to get off the hands, and the use of a barrier cream is advisable.

The setting time appears to be the same with this type of plaster as with the ordinary bandages supplied by the same firm, but hard setting is much quicker.

Examples. *Below-knee walking plaster*—Three six-inch bandages and one and a half four-inch bandages give a strong plaster for a man weighing 11 stones (154 pounds) and will last the necessary period of six to eight weeks. The weight is two pounds as against three pounds when ordinary plaster (five six-inch and two four-inch bandages) is used.

Knee cylinder for adult (groin to ankle)—Five six-inch bandages were used as against nine to ten with ordinary plaster-of-Paris.

Scaphoid plaster—Two and a half four-inch bandages gave a strong plaster which lasted four weeks. With ordinary plaster three and a half four-inch bandages are required to produce a splint of similar strength for a working man.

Jackets and spicas—A limited experience with resin plasters for plaster jackets and a shoulder spica proved very satisfactory. Fewer bandages were necessary and the final plaster was durable and strong. The plaster jacket required six to eight six-inch bandages.

We have a limited experience of using orthodox commercial plaster-of-Paris bandages dipped into a mixture of resin, but the preparation of the mixture was troublesome and time-consuming. One member of the nursing staff developed a dermatitis of the hands and forearms which is slow in subsiding after two months. We have experienced no skin reactions from the plaster-of-Paris resin bandages, although the use of a barrier cream is advisable both to facilitate the removal of the dry plaster and to minimise the risk of a dermatitis.

SUMMARY

A short experience with melamine resin plaster-of-Paris bandages is described. The bandages make a lighter splint with a saving of material and time, and the splint is more durable.

* The plaster bandages were kindly supplied by Messrs Smith & Nephew Ltd., of Hull, England, makers of the Gypsona plaster bandage.

EXPERIMENTAL PLASTER BANDAGES

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This brief report is submitted by one who has used plaster-of-Paris for twenty-five years, has experimented with other forms of splintage, but prefers something that sets with extreme rapidity. It is realised that some surgeons prefer a plaster that sets rather slowly.

From time to time attempts are made to produce a type of tailor-made external splintage that is better than plaster-of-Paris. External splintage is used in two rather different ways. The first is for short-term splintage of fractures or joints where the splint must set rapidly to ensure that the position obtained by manipulation is retained. The second type is where some form of splintage is required for long periods and it is of advantage that it should be light, durable, unaffected by water and permeable to x-rays. It is not so important that the second type of splint should take a long time to make and become hard.

For the first type, plaster-of-Paris has been used for a very long time. The advantages of plaster-of-Paris are: ease of application, rapidity of application, rapidity of setting and comparative cheapness. The disadvantages are: relatively heavy weight, relative impermeability to x-rays, failure to stand up to unprotected weight bearing when applied to the lower limb and rapid softening and loss of efficiency if moistened.

So far, attempts to replace plaster-of-Paris have meant that, while the advantages of lightness, durability and permeability to x-rays were obtained, there was difficulty in application. Rather special skill was required for the application of substances like glass acetate fibre. These also took a long time to set and were therefore of little value in a busy clinic or where fractures had been reduced. Other methods have required the use of heat ovens and a special technique in application as well as a good deal of time, and for this reason they are inapplicable to a busy clinic.

There have been suggestions that by incorporating substances such as resins with plaster-of-Paris, the advantages of lightness and greater strength would be obtained. An experimental resin-impregnated plaster has recently been on trial at this group of hospitals, and it appears to possess advantages and no disadvantages. The bandages are soaked in water in the normal manner, and the plaster sets rapidly. It is durable. It is non-irritating to the skin and it has the additional advantages of being lighter and stronger. Only two-thirds of the normal amount of bandages are required.

The dry bandage looks like any other plaster bandage. It feels perhaps a little rougher. In soaking, hardly any of the plaster is lost in the water. Whereas three orthodox plaster bandages left 11·5 grammes (dried) of plaster in the bucket, those of the new type left only 0·2 gramme. The bandage could be wrung out almost dry after soaking.

The new bandages were economical in use. For example, whereas with ordinary plaster I would use one six-inch bandage in the form of a slab fixed with an open-wove bandage to immobilise a Colles's fracture, with the new type only one four-inch bandage is required and, if anything, the new splint is stronger than the old one. A below-knee plaster for a man can be made with two and a half six-inch bandages. There is no skin irritation when the plaster bandage is applied directly to the skin.

Tests of strength were carried out. One six-inch plaster bandage of orthodox type was made into a slab fifteen inches long and allowed to set and dry. A similar slab was made from the resin-impregnated plaster bandage. The slab was supported between wooden blocks and weights were applied until it cracked. The weights required to cause a crack were: orthodox plaster, three and a half pounds; resin-impregnated plaster, four and a half pounds.

This is admittedly a crude method of testing because a flat slab is nothing like so strong as a curved slab or cylindrical plaster normally applied, but it nevertheless gives an indication of the added strength imparted by the resin.

Three ordinary four-inch bandages after soaking and drying weighed 336 grammes, whereas three new bandages similarly treated weighed 333 grammes, which suggests that they are very slightly lighter than the old ones.

SUMMARY

Clinical trials and experiments with a new plaster-of-Paris bandage which can be used in the normal manner are described. Only about two-thirds of the usual number of bandages are required to produce a plaster case of equivalent strength.

Supplies of this new resin-impregnated plaster bandage were kindly provided by Messrs Smith & Nephew Ltd., of Hull, England, makers of the Gypsona plaster bandage.

TRANSPLANTATION OF EPIPHYSIAL CARTILAGE

An Experimental Study

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Clinical observations upon the fate of reimplanted or transplanted epiphysial cartilage are few, and an examination of the literature reveals only two cases in which this has been successfully achieved. In 1949 Key described a case in which a boy of eleven years sustained a fracture of the proximal end of the radius. The fracture line passed distal to the epiphysial cartilage, and the head and the neck of the bone were removed surgically and replaced in their normal position. Four years later no shortening was apparent. This case is not, however, completely convincing. The radiographs do not show clearly that the epiphysial cartilage has survived, and, since the proximal end of the radius contributes little to the growth of the radius at this age, the failure of the epiphysial cartilage to grow may not have been revealed in this study. On general grounds, however, one might expect such a reimplanted cartilage to survive. Helferich (1899), Heller (1914), Fohl (1929), Sousa Pereira (1937) and Ring (1955*b*) have all investigated this point in the experimental animal. Their results suggest that after excision and reimplantation in its normal position the epiphysial cartilage survives and plays its normal part in growth. In this type of experimental work it appears that successful reimplantation is more common if the epiphysial cartilage is removed with a very thin sliver of the adjacent bone.

The fate of an autogenous transplant of epiphysial cartilage was described by Straub in 1929. His patient was a boy in whom the distal end of the tibia sequestrated after an attack of osteomyelitis at the age of four years. Two years later a strip of bone including part of the distal epiphysial cartilage was removed from the normal medial malleolus and implanted to fill the defect on the other side. At the age of twenty the boy was found to have 5 centimetres of shortening in the tibia compared with 4·5 centimetres at the time of operation. This example of an apparently successful transplant of epiphysial cartilage is open to two main criticisms: the destruction of the original epiphysial cartilage may have been incomplete, or the growth in the tibia may have occurred by an increase in the contribution from the upper end. The procedure adopted in Straub's case has a very limited clinical application since there is no readily available source of epiphysial cartilage, and the dangers of interfering with the normal epiphysial cartilage are well known. Autogenous grafting of non-epiphysial cartilage is a well established procedure in certain other sites, however (Gillies 1943, 1948), and the fate of such grafts has been studied histologically by Peer (1939, 1940). There can be no doubt that this tissue survives for long periods after autogenous grafting. The cartilage cells remain viable, and Dupertuis (1950) has recorded growth in autogenous cartilage grafts in young subjects. The fate of the homogenous graft is less clear. Many authors believe that the cartilage cells persist and that for long periods the matrix remains unchanged, although in most cases there is a slow replacement by fibrous tissue or bone (Schofield 1953). The absence of an immunity response after such homogenous grafts may be related to the absence of blood vessels, since experiments with other tissues show that an immunity response does not develop if blood vessels from the host fail to penetrate the graft.

From an experimental viewpoint transplantation of epiphysial cartilage affords a method of studying the functional survival of a cartilage graft. Urist and McLean (1952) transplanted epiphysial cartilage to the anterior chamber of the eye. Results from both autogenous and

homogenous transplants were similar: in the early stages new bone was produced, but after seven weeks there was no evidence of proliferation of cartilage cells. Lacroix (1951) placed small pieces of epiphysial cartilage from one rabbit beneath the renal capsule of a litter mate. Growth for the first three weeks proceeded normally with the formation of a bony epiphysis and metaphysis, but after this period growth in length of the specimen was slight. This is a common phenomenon in the homogenous transplantation of many tissues, and it suggests a specific immunity on the part of the host. This immunity may, however, be associated with the abnormal site of the transplant, and may thus be organ-specific or tissue-specific. Such a factor can be eliminated if the epiphysial cartilage be transplanted into the end of a long bone. Experiments of this type were reported by German authors in the early part of this century. In general their experiments involved transplantation of large masses of bone, often the whole bony epiphysis, the epiphysial cartilage and the metaphysis. Such grafts failed to survive homogenous transplantation (Heller 1917). Certain experiments (Rehn and Wakabayashi 1912, Tappeiner 1915) are invalid since they were performed upon the elbow, a region in which little growth occurs, and in which retardation may be obscured by overgrowth at the other end of the bones involved. Among the more recent observations, those of Silfverskiöld (1934) are the most detailed. In the rabbit he found that autogenous exchange of the distal ulnar epiphysial cartilage between the forelimbs produced cessation of growth in six of eleven animals, and considerable retardation in the rest. Homogenous transplantation of the ulnar epiphysial cartilage into the same limb of a litter mate produced a complete cessation of growth.

The interpretation of experimental studies of this type needs considerable caution. It is possible for growth to appear more or less normal, due not to the survival of a graft, but from regeneration of epiphysial cartilage, for it has been shown (Ring 1955*a*) that under favourable conditions epiphysial cartilage in the young rabbit can re-form after complete excision. Failure of growth, on the other hand, may be due to an immunity reaction in the host, or to technical errors. Free transplants of epiphysial cartilage must unite on both epiphysial and diaphysial surfaces with the host bone, and the epiphysial cartilage itself must become revascularised. In the present series of experiments an attempt has been made to control some of the many variable factors in transplantation by comparing a group of animals receiving autogenous transplants with a group receiving homogenous grafts.

METHOD

Young rabbits aged from three to six weeks were used. Each animal was anaesthetised with ether, and the distal end of the left ulna was exposed by a longitudinal incision. The tendons were retracted from the bone, which was freed for most of its circumference. Parallel knife cuts were now made transversely through the bony epiphysis and metaphysis. The isolated cartilage, with its thin attached slivers of bone, was then gently freed from the radius and removed together with its surrounding perichondrium. In one group of animals a similar operation was performed upon the right ulna, and into the defect in the ulna was placed the epiphysial cartilage from the left side. The operation was completed in this group by the reinsertion of the right epiphysial cartilage into the left ulna. Care was taken to orientate the epiphysial cartilage with its normal relation to epiphysis and metaphysis, but it proved impossible to get a consistently good fit in this autogenous transplant, particularly since the excised cartilage is the mirror image of the one it replaces.

Animals undergoing homogenous transplantation were all litter mates, but were not pure bred, and no attempt was made to sex donor and recipient. Animals were anaesthetised in pairs, and by a technique similar to the one described above the left ulnar epiphysial cartilage was excised from one animal and transplanted into the other. Here again, a perfect fit in the transplant was seldom obtained, mainly because the animals, although of the same age, varied considerably in size.

Immediately after operation the animals were radiographed, and the forelimbs were protected by a loose dressing of adhesive tape. This tape was removed fourteen days after operation, when the animals moved freely, using the operated limb without difficulty or obvious disability.

TABLE I

GROWTH IN MILLIMETRES OF TEN NORMAL RABBITS DURING FIRST TWENTY-FIVE WEEKS OF LIFE

Week of life	Rabbit Number										Total	Mean
	113	123	124	125	127	131	130	141	75	132		
4	4·5	4	4·75	4·5	4·75	5	4	5	2·5	4	43	4·3
5	4·5	4	4·75	4·5	4·75	5	4	4·5	2·5	4	42·5	4·3
6	4·5	2·75	2·5	3·25	2·75	4	4·25	3·5	2	3·25	32·75	3·3
7	2·8	2·75	2·5	3·25	2·75	4	4·25	4	1·6	3·25	31·15	3·1
8	2·8	2·75	3·25	2·5	2	3·25	3	4	1·6	2·75	27·9	2·8
9	1·25	2·75	3·25	2·5	2	3·25	3	2	1·6	2·75	24·35	2·4
10	1·25	2·5	3	2·25	3	2·5	1·25	2	3	3	23·75	2·4
11	1·75	2·5	3	2·25	3	2·5	1·25	2	3	3	24·25	2·4
12	1·75	1·25	1·5	1·5	1·25	1·5	2·75	2	2·5	1·75	17·75	1·8
13	2·25	1·25	1·5	1·5	1·25	1·5	2·75	2	2·5	1·75	18·25	1·8
14	2·25	2·25	2	2	2·25	2·5	2·25	2	2·25	3	22·75	2·3
15	1·5	2·25	2	2	2·25	2·5	2·25	1·25	2·25	3	21·25	2·1
16	1·5	0·5	1·25	1	1	2	1·75	1·25	2	1·75	14	1·4
17	1·25	0·5	1·25	1	1	2	1·75	0·25	2	1·75	12·75	1·3
18	1·25	1	1·25	1	1·25	1	0·75	0·25	2·5	1·5	11·75	1·2
19	1·5	1	1·25	1	1·25	1	0·75	1·5	2·5	1·5	13·25	1·3
20	1·5	0·25	1·5	0·75	0·5	0·5	0·75	1·5	0·5	—	7·75	0·9
21	1	0·25	1·5	0·75	0·5	0·5	0·75	0	0·5	—	5·75	0·6
22	1	—	0·75	0·5	0·75	1	0·75	0	1	—	5·75	0·7
23	0·5	—	0·75	0·5	0·75	1	0·75	0	1	—	5·25	0·7
24	0·5	—	0·25	—	0·5	0·25	—	0	0·5	—	2	0·3
25	—	—	0·25	—	0·5	0·25	—	0	0·5	—	1·5	0·3

— = not measured.

Radiographs were repeated every two weeks during the period of study, which in some animals continued into adult life. The animals were lightly anaesthetised, and were radiographed in the prone position with each forelimb flexed at the elbow and with the paw outstretched. This position gave a standard view of the radius and ulna, and with a tube-film distance of 100 centimetres it was shown that the errors in this technique from distortion were less than 0·5 millimetre. Some animals were killed during the period of growth and provided material for histological study. The forelimbs were removed, fixed in Susa and afterwards decalcified in 5 per cent nitric acid. A sample series of sections was taken longitudinally through the distal end of the ulna and stained with haematoxylin and eosin.

OBSERVATIONS

AUTOGENOUS TRANSPLANTATION

Radiological—In order to provide a control series for this group of animals the growth rate of the ulna of a number of normal rabbits was determined. These rabbits were radiographed at intervals of one, two or three weeks during the whole period of growth. The weekly growth rate was calculated by dividing the increments of growth in the ulna by the interval between radiographs. The results of this calculation may be seen in Table I, in which the mean growth rate of ten rabbits is recorded.

TABLE II

GROWTH OF ULNAE AFTER TRANSPOSITION OF DISTAL EPIPHYSIAL CARTILAGES

Rabbit number	Age at operation (*weeks*)	Age at death (*weeks*)	Duration of experiment (*weeks*)	Growth		Predicted growth (*millimetres*)
				Left (*millimetres*)	Right (*millimetres*)	
241	3	4·5	1·5	1	1·5	6·5
157	4	5·5	1·5	3	3	6
308	5	7	2	5·5	5·5	6·5
311	5	7	2	4·5	3·5	6·5
310	5	8	3	5	5·5	9
354	3	7	4	8	12	15
172	4	9	5	9	10	16
170	3	10	7	19*	14	22·5
360	4	13	9	20*	17·5	24·5
156	4	15	11	10	11·5	28·5
351	4	16	12	11	21·5	30
236	3	15	12	17	18	33
309	5	18	13	33*	18	28·5
171	3	20	17	15	23	39
319	3	22	19	34*	36*	40·5

Growth is predicted to nearest 0·5 millimetre.
* Successful transposition.

The mean growth rate was used to predict the amount of growth to be expected in the ulnae of the experimental animals. Such a method may, of course, be very inaccurate (particularly when dealing with rabbits from either very small or very large mothers) and must be used in conjunction with other observations. It was soon apparent that measurements during the first few post-operative weeks gave little indication of the fate of the transposed cartilage. The firm attachment of the distal end of the radius to the distal epiphysis of the ulna causes the fragments of the latter to be distracted, and in the early post-operative days an apparent lengthening of the ulna may be due to stretching of the fibrous tissue between the graft and the host bone, rather than to active growth within the graft. This same firm attachment would also produce a considerable bowing of the forelimb if the epiphysis and diaphysis of the ulna later became more firmly united. The radius being fixed at each end by its attachments to the ulna and to the elbow, its growth is dependent upon growth

of the ulna. When the latter fails to grow the curvature of the radius increases and a valgus deformity of the paw ensues. When this distortion of the radius became marked the ulna was also bowed and measurements of its length under these conditions were inaccurate.

In Table II the growth of the ulna in the experimental animal is compared with the predicted growth. In only one animal was growth of the ulna greater than predicted. In the others the apparent shortening of the experimental group varied greatly. Using this method of prediction wide variations might indeed be expected, but if growth of the ulna is normal these variations should be scattered evenly above and below the predicted figure. Low measurements in the experimental animal may be due either to deficient growth of the transposed cartilage or to the inclusion of a large number of technical failures within this group. With this in mind the animals were re-examined to determine the success or failure of each transposition on grounds other than measurements of the final length. Transposition was

FIG. 1

Serial radiographs at monthly intervals of the left forelimb of R.309. Growth is proceeding normally after transposition of the epiphysial cartilage of the ulna.

deemed successful if the radiographs showed the following features during the period of growth: 1) the presence of a band of radiotranslucency corresponding to the normal epiphysial cartilage; 2) clouding of the metaphysis indicating a normally active epiphysial cartilage; 3) a normally curved radius; and 4) a steady increase in the length of the ulna.

In practice it was impossible to predict the fate of a transposition within the first four weeks after operation, and this re-examination was confined to the older animals. Nine animals, representing eighteen transpositions, fell into this group. Of these eighteen transpositions, only five could be assessed as completely successful, and these animals are indicated by an asterisk in Table II. They satisfy the criteria listed above, and in their ulnae growth has exceeded 75 per cent of that predicted (Fig. 1). In the other transpositions failure of growth often occurred after an apparently successful graft and was usually followed by bony fusion between epiphysis and diaphysis. This fusion occurred at almost any time after the operation, and there was no interval after which one could be certain that normal

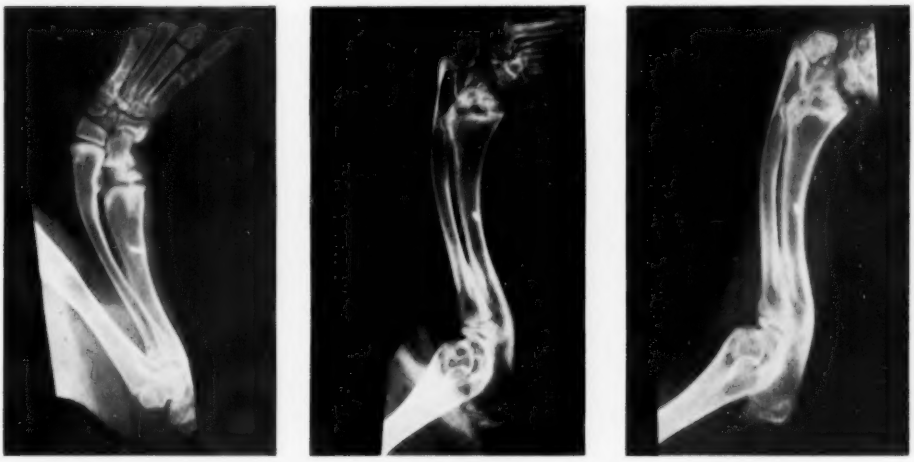

FIG. 2

Serial radiographs at monthly intervals of the right forelimb of R.309. The transposition has failed, the radius and ulna are curved, and the ulna is expanded at its distal end.

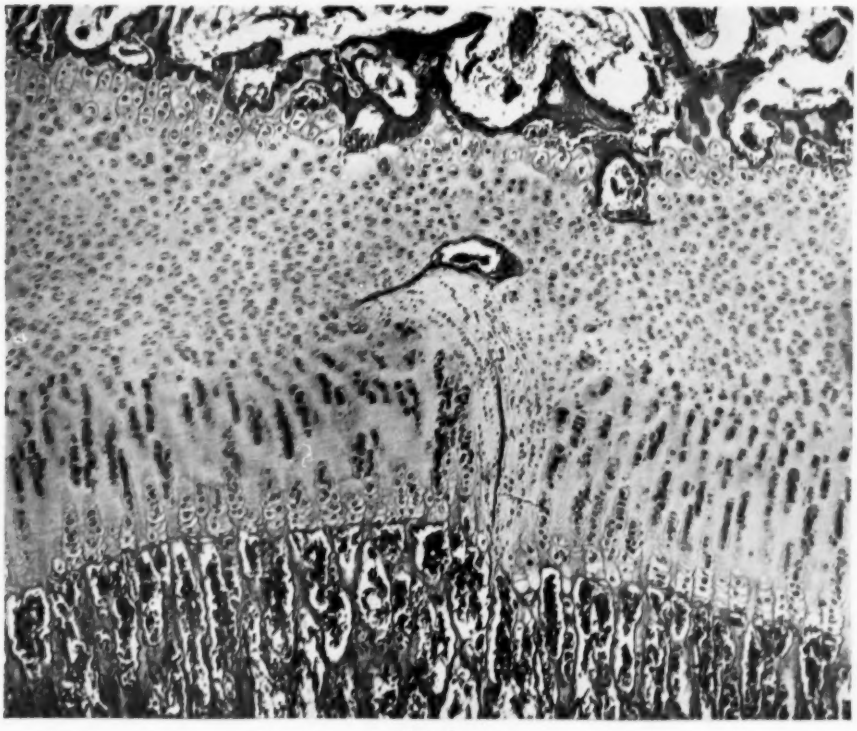

FIG. 3

The epiphysial cartilage two weeks after transposition. The graft has united with the host bone, and apart from a little central fibrosis appears normal. R.157 (*right*). (×80.)

growth would occur, although the chance of a successful transplant increased the longer it survived without radiographic evidence of deterioration. One of three main radiographic changes was associated with failure to grow: 1) complete disorganisation within the grafted area; 2) calcification within the epiphysial cartilage; and 3) narrowing of the epiphysial cartilage. Each of these changes progressed to give fusion of epiphysis and diaphysis (Fig. 2).

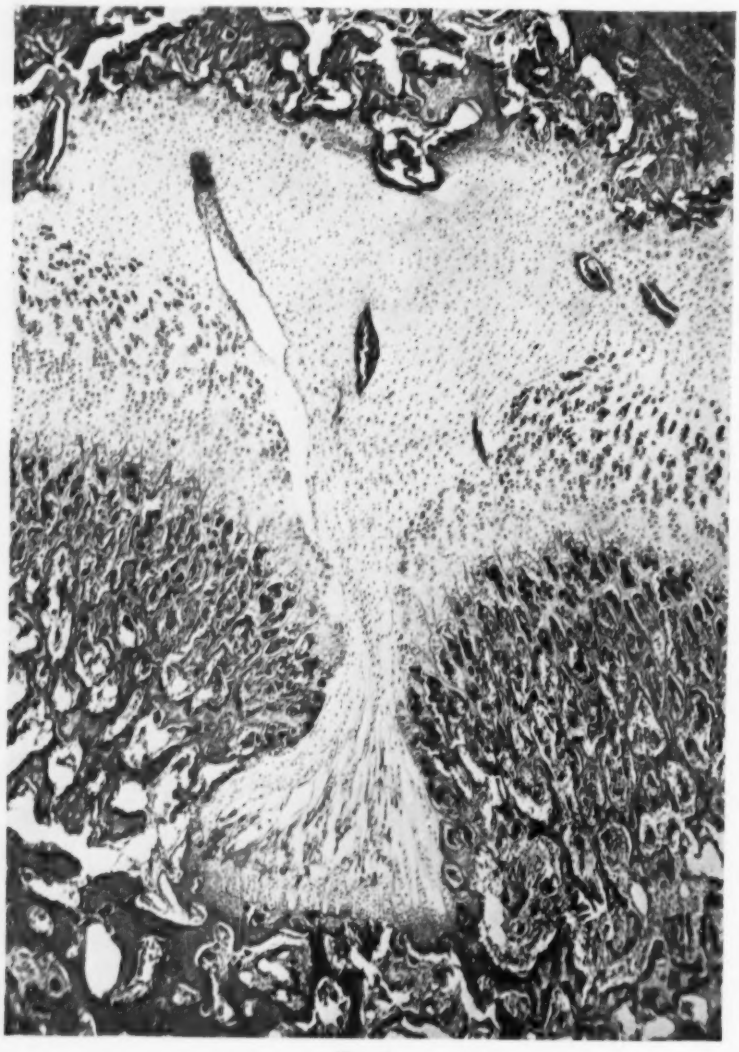

FIG. 4

The epiphysial cartilage two weeks after transposition. The graft shows considerable central necrosis, with failure of metaphysial invasion. R.311 (*right*). (× 45.)

Histological—Sections from these animals showed that the transposed cartilage united rapidly with the metaphysial side of the host bone, and somewhat less rapidly with the epiphysial side (Fig. 3). Between the latter bone and the graft, cartilage cells were often visible in the immediate post-operative period, a common feature in the repair of any bony

defect in the young animal. The grafted epiphysial cartilage showed no change until the third post-operative week, when the central cartilage cells became eosinophilic, and the surrounding cartilage matrix became replaced by fibrous tissue (Fig. 4). The cellular degeneration was followed by calcification of the degenerated central cartilage, and subsequently by a variable amount of bony replacement. This new bone formation united the epiphysis and metaphysis. In some animals the bridge was very narrow (Fig. 5). Such a bridge, if central, may be no barrier to bone growth, since the area of metaphysis on its proximal aspect is subject to continual encroachment by the bone marrow. Animals in which the bridge was narrow showed in general good post-operative growth. When the bridge was wide, presumably following widespread central cartilage degeneration, the epiphysial cartilage

FIG. 5

A small central bony area seven weeks after transposition. R.170 (*left*). (× 125.)

persisted only as a narrow ring around this area of new bone formation (Fig. 6). In these animals shortening was considerable.

HOMOGENOUS TRANSPLANTATION

Radiological—Transplantation of the left distal ulnar epiphysial cartilage was performed in thirteen pairs of litter mates giving a total of twenty-six experimental ulnae. The survival of these animals and their post-operative growth is recorded in Table III.

From these figures it is apparent that in no animal has the growth on the side operated upon equalled or exceeded that of the control. Using the criteria for successful transplantation listed previously the twenty-four animals surviving more than four weeks were subjected to

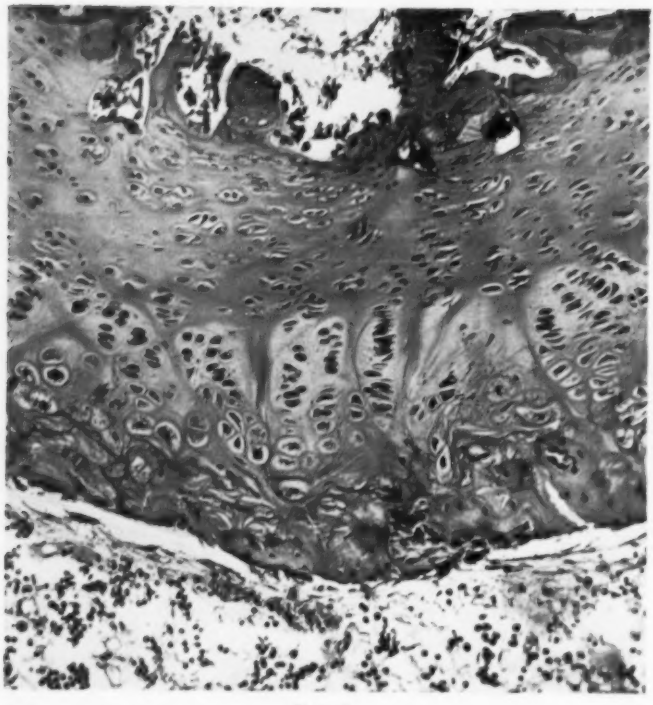

FIG. 6

Part of the peripheral ring of epiphysial cartilage remaining after extensive central bony replacement ten weeks after transposition. R.156 (*right*). (×150.)

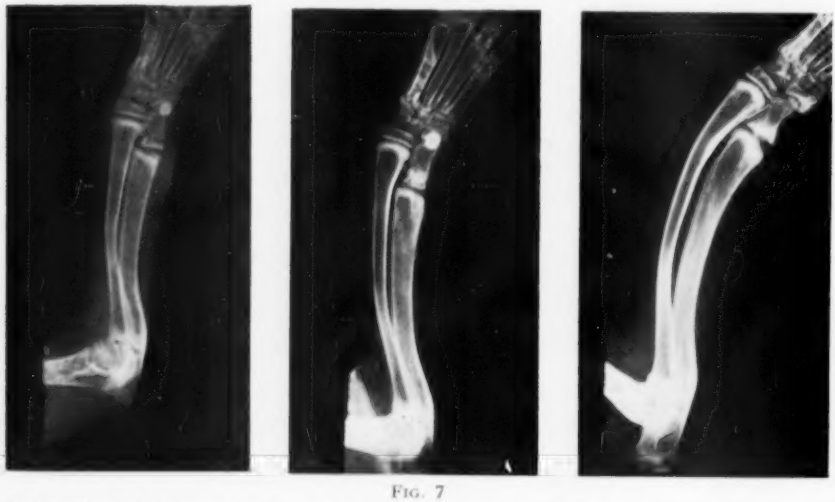

FIG. 7

Serial radiographs at monthly intervals of the forelimb of R.337. Although the epiphysial cartilage remains the ulna is considerably shorter than the control.

re-examination. In only three animals was the radiographic appearance of the distal end of the ulna compatible with normal growth, and in each of these the epiphysial cartilage

TABLE III

GROWTH OF ULNA AFTER HOMOGENOUS TRANSPLANTATION OF DISTAL EPIPHYSIAL CARTILAGE

Rabbit number	Age at operation (*weeks*)	Age at death (*weeks*)	Duration of experiment (*weeks*)	Growth	
				Experimental side (*millimetres*)	Control side (*millimetres*)
88	4	25	21	21	42
89	4	17	13	8·5	34
101	4	24	20	12	36
102	4	23	19	10·5	38·5
132	4	20	16	13	40·5
133	4	9	5	10·5	20
134	4	9	5	10	19·5
135	4	9	5	9·5	15·5
136	4	15	11	16	28
137	3	9	6	10	17·5
149	4	10	6	9·5	12
151	4	10	6	13·5	17·5
154	4	12	8	11	16
155	4	11	7	7·5	14
168	3	10	7	12·5	20·5
169	3	16	13	15	35
320	3	21	18	26·5	43*
321	3	21	18	20	37·5
326	4	21	17	18·5	31
327	4	5·5	1·5	3·5	3·5
333	4	18	14	15·5	30·5
334	4	18	14	15·5	31·5
335	4	16	12	13	25
336	4	8	4	7	9·5
337	4	18	14	19	29*
338	4	18	14	12	17·5*

* Animals in which final radiographs show persistence of narrow epiphysial cartilage.

was narrow (Fig. 7). In none of these three animals was the growth of the experimental limb within 75 per cent of the control (Table III).

Examination of the serial radiographs of the animals in which transplantation was performed suggests that growth from the grafted epiphysial cartilage at first proceeded

normally, even before bony union had occurred between the thin slivers of bone at each end of the transplant and the bone of the host. The size of the epiphysial cartilage indicated by the area of radiotranslucency often increased, possibly from oedema of the cartilage. The broadening of the epiphysial cartilage was followed by a progressive narrowing, sometimes proceeding to obliteration of the epiphysial cartilage by bony fusion. This narrowing was normally apparent by the fifth or sixth week. In some animals small specks of calcification appeared within the epiphysial cartilage, often as early as three weeks after operation, and in one animal only ten days after grafting. In other rabbits absorption in the region of the metaphysis occurred and was followed by marked bowing of the ulna.

Histological—Sections from these animals were in many respects similar to those of the

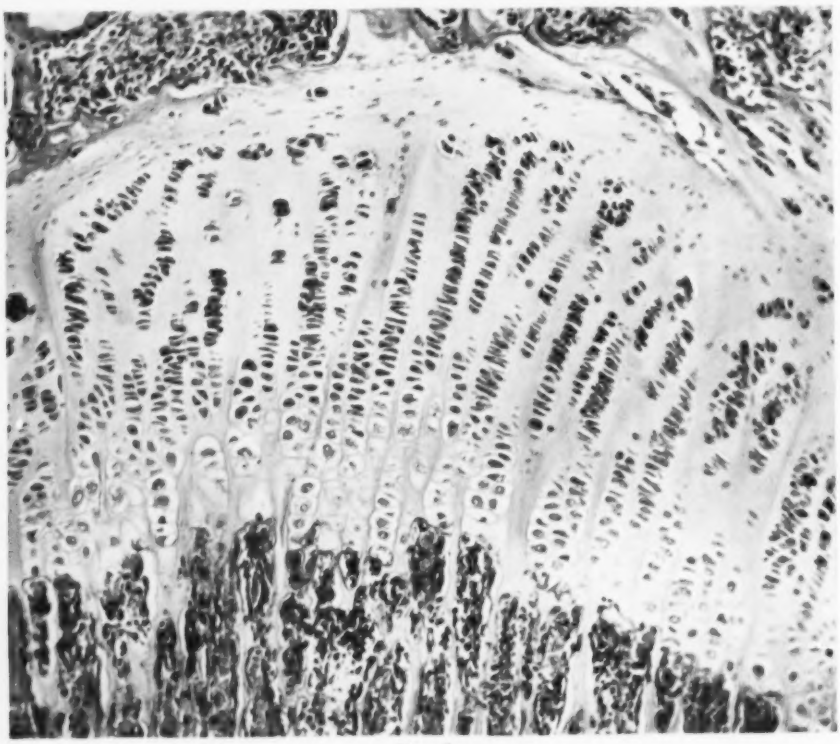

FIG. 8

The epiphysial cartilage four weeks after homogenous grafting. The reserve zone has degenerated, the remaining cartilage is intact, and the metaphysis still appears active. R.336. (×135.)

transposed group. Most animals showed some central cartilage necrosis and this proceeded to a variable amount of bony union between the centre of the epiphysis and the metaphysis of the host. Within the reserve zone, however, certain other changes became apparent. During the first three weeks the structure of the cells in this region was apparently unchanged, although there was no evidence of recent cellular division. After this period the cells degenerated completely, and the reserve zone was replaced by a transversely running band of fibrous tissue (Fig. 8). The cartilage columns persisted unchanged, apart from a little longitudinally running fibrous tissue. The metaphysis was relatively avascular, its columns of new bone were very short, and osteoblasts were few in number. This thin, inactive, epiphysial cartilage was found in specimens for many weeks after operation, and complete bony union between epiphysis and metaphysis was uncommon.

DISCUSSION

The measurements in the Tables record the total growth of the ulna, and no attempt has been made to divide this into components from the distal and from the proximal ends. From previous experiments upon the ulna of the rabbit in which lead markers were inserted into the shaft of the bone it was found that the distal epiphysial cartilage contributed about 85 per cent of the post-natal growth of the bone. The remainder of the growth of the bone is due to growth in the proximal epiphysial plate, and to the enlargement of the proximal end of the ulna by appositional growth. The rate of growth at the proximal end is occasionally increased if the distal epiphysial cartilage is destroyed, but this increase is only of the order of one or two millimetres during the whole growing period. The growth of the proximal end of the ulna approaches a millimetre in three weeks, and this figure accounts for the slow increase in length seen in the experimental ulna when the distal epiphysial cartilage is clearly inactive.

It is apparent from the Tables that failure of a transplanted epiphysial cartilage to survive is not immediately followed by a cessation of growth. Some of this growth is undoubtedly due to distraction in the region of the operative intervention by the growing radius, but in many radiographs it is clear that growth of the transplant continues without interruption in its new site, although often at a slightly slower pace. In most animals growth of the graft eventually ceases and may be followed by fusion between epiphysis and diaphysis.

Failure of a graft to grow may be due either to technical or to immunological factors. It is difficult to arrange for the bed of the host to fit accurately the grafted area, and in some cases bad seating of the graft may have been responsible for delay in union between its ends and the host bone. Gross infection was not observed within this series, but minor infection may well have played a part in some failures. Most of the technical difficulties were probably associated with vascular changes, a feature discussed more fully below.

Since the proportion of failures was high it is important to determine the fate of the epiphysial cartilage if it is isolated and excised in the manner described and then reimplanted at the same site. In a recent study of this type it was found that of nine animals followed for five weeks or more, six showed little or no shortening and three were failures (Ring 1955*b*). Transposition between the forelimbs is technically more difficult, but the failure of thirteen transpositions out of eighteen in the present study is perhaps a greater increase than might be expected. The even greater failure rate in the homogenous transplantations can hardly be due to an increase in technical difficulties alone, although it is difficult to obtain animals matching well in size. From the radiographs alone, however, no direct evidence of the introduction of an immunity factor is forthcoming.

From the measurements upon the radiographs the mean post-operative growth in both autogenous and homogenous transplants can be assessed. A direct comparison of Tables II and III gives the following results (Table IV).

TABLE IV

COMPARISON OF GROWTH AFTER AUTOGENOUS AND AFTER HOMOGENOUS TRANSPLANTS

Group	Duration of experiments (*weeks*)	Growth	
		Experimental side (*millimetres*)	Control side (*millimetres*)
Left limb from autogenous group	8	13	20·8
Right limb from autogenous group	8	13·3	20·8
Homogenous transplant . .	11·5	13·1	25·6

Although it appears that growth in each of these groups is similar, that recorded for the animals undergoing homogenous transplantation covers a much longer period. In Table V direct comparisons have been made between individual animals surviving for comparable

TABLE V

A Direct Comparison of Autogenous and Homogenous Grafts Surviving Similar Periods

Autogenous grafts					Homogenous grafts			
Animal number	Duration of experiment (*weeks*)	Growth		Predicted growth (*millimetres*)	Animal number	Duration of experiment (*weeks*)	Growth	
		Left (*millimetres*)	Right (*millimetres*)				Experimental side (*millimetres*)	Control side (*millimetres*)
354	4	8	12	15	336	4	7	9·5
172	5	9	10	16	134	5	10	19·5
170	7	19	14	22·5	155	7	7·5	14
360	9	20	17·5	24·5	154	8	11	16
156	11	10	11·5	28·5	136	11	16	28
351	12	11	21·5	30	335	12	13	25
236	12	17	18	33	169	13	15	25
309	13	33	18	28·5	89	13	8·5	34
171	17	15	23	39	326	17	18·5	31
319	19	34	36	40·5	102	19	10·5	38·5
Total		176	181·5	277·5			117	240·5
Mean		17·6	18·2	27·8			11·7	24·1

periods of time. All the animals undergoing autogenous transplantation and surviving more than three weeks are included and are matched with animals surviving for a similar period in the homogenous group.

Direct comparison of individual limb growth rates shows that in fourteen of twenty limbs the autogenous group exceeded that of the homogenous. Measurements of shortening gave the following results (Table VI).

TABLE VI

Shortening after Autogenous and Homogenous Transplantation

Autogenous		Homogenous
Left	Right	
10·2 millimetres	9·6 millimetres	12·4 millimetres

The wide variation in the growth of individual bones prevents further comparison, but it is clear that, under comparable conditions, the growth of the homogenous grafted cartilage is less active. Comparison of the histological changes in these two groups provides a clear explanation for the difference in the rate of growth. In both autogenous and homogenous transplants grafts may fail, if, after central degeneration of the epiphysial cartilage, bony union between epiphysis and diaphysis is sufficiently marked to provide a barrier to further growth. Ossification will then slowly spread through the cartilage and shortening will be

considerable. In the absence of such union however, growth after autogenous grafting proceeds normally, and the epiphysial cartilage shows every evidence histologically of normal activity. After homogenous grafting, however, degeneration of the reserve zone occurs (Figs. 9 and 10). This is the zone of active cartilage division upon which, in the end, all bone growth depends. When these cells fail to divide, growth in length ceases, although the less active area of the cartilage apparently persists unchanged. Subsequent lengthening of the ulna is due to distraction by the radius, and the growth in this group of animals is purely passive.

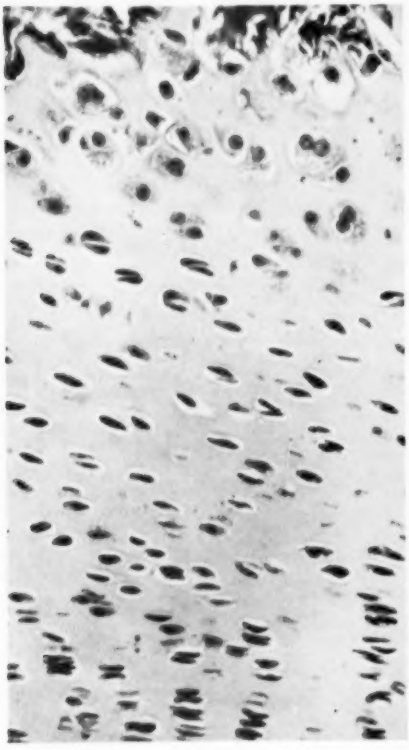

FIG. 9

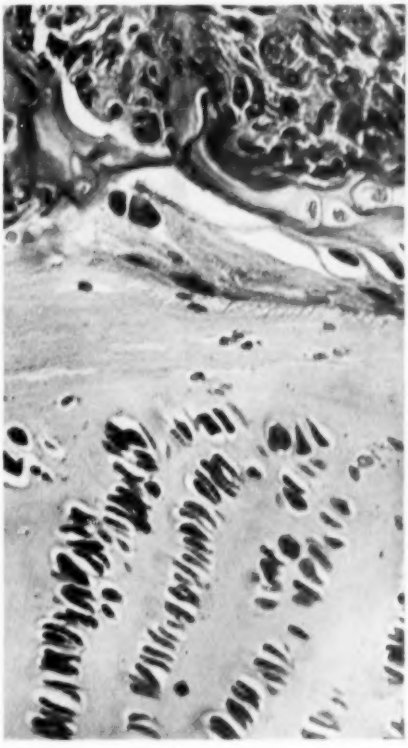

FIG. 10

Figure 9—The reserve zone three weeks after transposition. The cartilage cells are active. R.310. (×280.) Figure 10—The reserve zone four weeks after homogenous grafting. The cartilage cells have degenerated, although the columns appear intact. R.336. (×355.)

The central necrosis of the cartilage which is common to both autogenous and homogenous groups is probably due to a partial failure of revascularisation. It is clear from the observations of Haines (1933) and of Hurrell (1934) that the epiphysial cartilage is normally well vascularised. After transplantation the central area may be the last to be affected by the new ingrowth of vessels, and so may degenerate. From the sections in this study it is impossible to distinguish the relative vascularity of the different areas of the epiphysial cartilage, although it is clear that, after successful grafting, revascularisation does in fact occur.

Degeneration of the cells of the reserve zone after homogenous grafting is probably caused by an immunity reaction. These cells are actively dividing, and this area of the cartilage is normally supplied by blood vessels from the bony epiphysis. Revascularisation of the

transplant is thus followed by an immunity reaction similar to that seen after the grafting of other tissues. The relative freedom of non-epiphysial cartilage from this reaction is due to its avascularity, and possibly also to its low cell content. Since the cartilage columns contain dead cells, they will not provoke an immunity reaction and will survive long periods. It is interesting to see that invasion by the metaphysis does not occur in the absence of a reserve zone, and examination of the histological appearance of the metaphysis provides a good indication of the rate of bone growth (Figs. 11 and 12).

From these experiments it appears that autogenous grafting of epiphysial cartilage may have a limited surgical application, although there is no ready source of cartilage in man, and the revascularisation of larger cartilage grafts may be difficult. There is no evidence

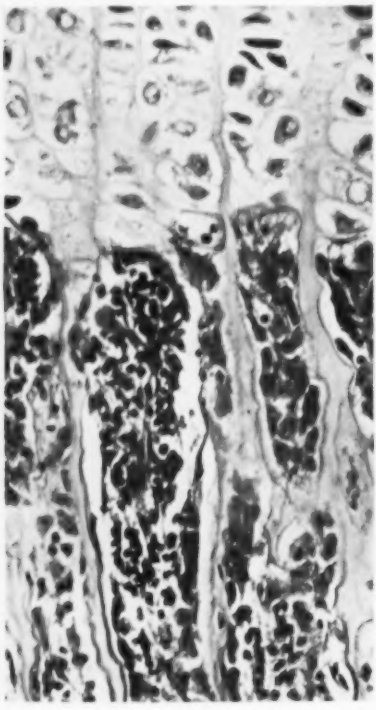

FIG. 11

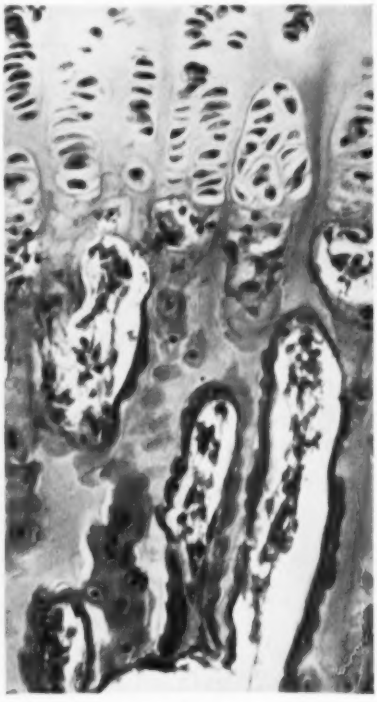

FIG. 12

Figure 11—An active metaphysis four weeks after homogenous grafting. The blood supply is good and many osteoblasts are present. R.336. (×300.) Figure 12—An inactive metaphysis seven weeks after homogenous grafting. Few osteoblasts are present, and the blood vessels are few. R.155. (×270.)

that homografts of epiphysial cartilage would be of any value, unless the barriers of transplantation immunity can be overcome.

SUMMARY

1. An experimental technique for the transplantation of epiphysial cartilage in the rabbit is described.

2. Autogenous transposition of the distal epiphysial cartilage of the ulna was followed by normal growth in five of eighteen animals.

3. Homogenous transplantation was unsuccessful in all the animals studied.

4. Homogenous grafting gives rise to an immunity reaction confined to the reserve zone of the cartilage.

5. It is suggested that the difference between the fate of homogenous grafts of epiphysial and non-epiphysial cartilage lies in the vascularity of the former.

I am indebted to Professor W. J. Hamilton for the facilities he has placed at my disposal and for his reading of the text. My thanks are also due to Mr R. J. McCulloch for his technical assistance. Mr E. V. F. Pittock is responsible for the photomicrographs.

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METABOLISM OF BONE SALT INVESTIGATED BY SIMULTANEOUS ADMINISTRATION OF ^{45}Ca AND ^{32}P TO RATS

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Within a short time after the parenteral administration of radiocalcium or radiophosphorus to animals the principal part of the dose is located in the skeleton. This uptake may be due to reversible processes (for instance, surface exchange) or to irreversible processes, or to both. Carlsson (1952) demonstrated that one day and onwards after the administration of radiocalcium to growing rats practically all of the activity recovered from the skeleton had been irreversibly deposited with the bone salt formed during the time of the experiment. Carlsson also showed how it is possible to determine the rate of formation of calcium salt in the incisor teeth from isotope data. Using Carlsson's technique, Bauer (1954*a*) determined the rate of formation of bone salt both in fractured and in intact bone shafts in adult rats.

The experiment to be described here was undertaken to investigate whether phosphorus is irreversibly incorporated in the skeleton fast enough to permit a calculation from isotope data of this rate. Radiocalcium and radiophosphorus were therefore administered simultaneously to rats, and the rate of bone salt deposition in various skeletal samples was calculated from the two sets of isotope data. It was found that there was no essential difference in the metabolism of the calcium and of the phosphorus of the bone salt.

EXPERIMENT

The series consisted of forty-three three-months old rats of both sexes from an inbred strain. Each rat received a subcutaneous injection of about 15 microC of ^{45}Ca and 30 microC of ^{32}P in an aqueous solution containing negligible quantities of calcium and phosphorus. The rats were killed by bleeding under ether anaesthesia in groups of three to five at intervals after injection ranging from one to 120 hours. The blood from each rat was collected and centrifuged. One millilitre of serum from each animal was mixed with 2 millilitres 15 wt per cent trichloroacetic acid. This mixture was centrifuged, and the clear residue was used for determinations of ^{32}P activity, and of total P according to the colorimetric method of Youngburg and Youngburg (1930). The unprecipitated serum from each group was pooled and the serum Ca was determined for each group according to the method of Clark and Collip (1925). The ^{45}Ca activity was determined on the solutions of Ca obtained in this way after they had been titrated with potassium permanganate.

In each animal the tibiae and the incisor teeth were dissected free from adherent soft tissues. By means of a circular saw the fibulae were removed from the tibiae, and the tibiae were divided in ends and shafts. In each animal the pooled right and left tibial ends formed one sample, the tibial shafts formed one sample, and the four incisor teeth formed one sample. Each sample was ashed in an electrical muffle at about 400 degrees centigrade, the ash was weighed, and the ^{45}Ca and ^{32}P activity determined on a nitric acid solution of the ash. The technique followed was that of Carlsson (1951 and 1952), except that a ratemeter was used instead of a scaler (Bauer 1945*b*).

The activity of each sample was determined with and without an aluminium absorber, which absorbed all the ^{45}Ca radiation and 50 per cent of the ^{32}P radiation. The counts per second (c.p.s.) emanating from ^{45}Ca and ^{32}P respectively were calculated from the data thus

obtained. Standard solutions were made in 100 millilitres of nitric acid containing ^{45}Ca and ^{32}P in the same proportion as in the injected dose. By reference to them the c.p.s. values of the various samples were expressed as per cent of the administered dose of ^{45}Ca and of ^{32}P. The activity values recorded hereafter are expressed in this way.

RESULTS

The ^{45}Ca activity values of the tibial shafts and of the serum are recorded in Figure 1. The corresponding ^{32}P values are recorded in Figure 2. From the values shown in Figure 1 the rate of deposition of calcium in the tibial shafts has been calculated according to the following formula (Carlsson 1952, Bauer 1954*a*): $U = \frac{C}{T \times S}$ where U = calcium uptake in milligrams per hour; C = the percentage of the dose of radiocalcium recovered from the calcified tissue in question, T hours after the administration of the isotope; and S = the average specific activity of the serum calcium expressed as a percentage of the dose per milligram of calcium during the interval of time in question (O—T hours). (The expression S × T is represented by the area enclosed by the serum activity curve in Figure 1.)

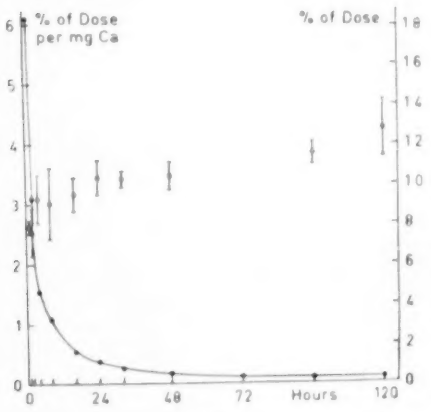

FIG. 1

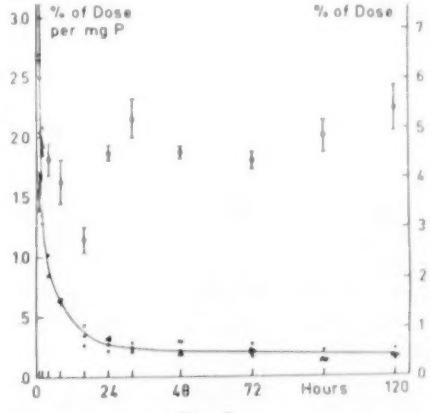

FIG. 2

Figure 1—*Specific activity of the serum Ca and the ^{45}Ca activity values of the tibial shafts at varying intervals after the simultaneous administration of ^{45}Ca and ^{32}P to young rats.* Specific activity of the serum Ca is plotted according to scale to the left of the graph. The values of the pooled serum of each group have been indicated in the graph. ^{45}Ca activity values of the tibial shafts are plotted according to scale to the right of the graph. The mean values are indicated ± the standard error. There were three animals in each of the 1, 2, 4, 8, 16 and 32 hour groups and five animals in each of the 24, 48, 72, 96 and 120 hour groups. Figure 2—*Specific activity of the serum P and the ^{32}P activity values of the tibial shafts at varying intervals of time after the simultaneous administration of ^{45}Ca and ^{32}P to young rats.* The specific activity of the serum P is plotted according to scale to the left of the graph. The individual values are indicated in the graph. ^{32}P activity values of the tibial shafts are plotted according to scale to the right of the graph. The mean values are indicated ± the standard error. The number of animals used was the same as that recorded in the legend to Figure 1.

It is seen from Figure 3 that the tibial shafts show a constant value of U from about twenty-four hours after the administration of the radiocalcium. This steady value represents a rate of deposition of calcium in the shafts of 0·03 milligrams of calcium per hour.

The U-values derived from the ^{32}P activity values in Figure 2 are recorded in Figure 4. It is seen that the steady value represents a rate of deposition of phosphorus in the shafts of 0·015 milligrams an hour.

The ratio of the Ca/P deposition rates in the tibial shafts was thus two to one.

DISCUSSION

Recent investigations have demonstrated that rapid, reversible processes seem to be of minor importance compared to skeletal growth for the redistribution of calcium and inorganic phosphorus in the skeleton. Thus isotope, once deposited in the bone salt, is released to the circulation only when the bone salt, containing the isotope, is reached by a resorption process which removes (dissolves) the bone salt together with the organic matrix (Carlsson 1951, 1952, Bauer 1954*a*, *b*, *c*, *d*).

By means of the above " U-formula " the rate of deposition of calcium in various parts of the skeleton of rats has been calculated from isotope experiments. However, during the

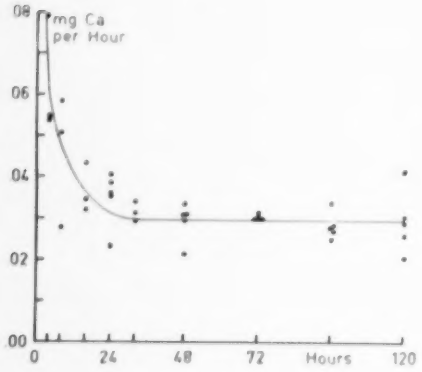

FIG. 3

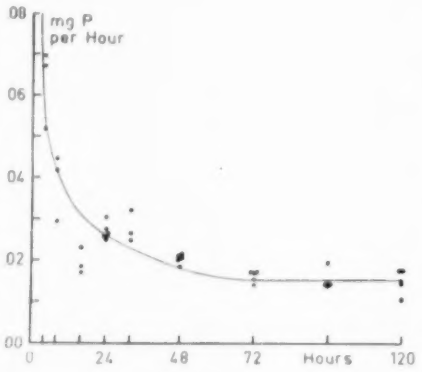

FIG. 4

Figure 3—*Calcium uptake in the tibial shafts as calculated from* ^{45}Ca *activity data after the simultaneous administration of* ^{45}Ca *and* ^{32}P *to young rats.* The calcium uptake has been calculated from the specific activity curve of the serum Ca and the tibial shaft ^{45}Ca activity values shown in Figure 1 by means of the " U-formula " (page 659). For lack of space the 1 and 2 hour groups have been omitted. The individual values are recorded in the graph. Figure 4—*Phosphorus uptake in the tibial shafts as calculated from* ^{32}P *activity data after the simultaneous administration of* ^{45}Ca *and* ^{32}P *to young rats.* Consult the note under Figure 3 for an explanation of the values recorded.

initial hours after the administration of the isotope the rate of deposition of calcium, arrived at by this method, is found to be higher than the subsequent steady rate (Fig. 3). The same is true for the phosphorus calculations (Fig. 4). The interval after the administration of the isotope during which this computation is influenced by reversible uptake processes is determined by the ratio of the exchangeable to the non-exchangeable fractions of the total activity uptake in a certain specimen of the skeleton. A more rapid fall in the serum specific activity respectively a lower ratio of the exchangeable fraction to the non-exchangeable fraction of the total activity uptake thus tend to decrease this initial time interval (Bauer 1954*c*). By comparison of the blood activity curves in Figures 1 and 2 it is seen that the calcium activity falls faster than does the phosphorus activity. Practically all the body calcium is located in the bone salt, whereas a considerable part of the body phosphorus is located outside the bone salt. The period after the administration of the isotopes during which the reversible processes might be expected to interfere with the calculation of the rate of the irreversible deposition process is thus longer in the ^{32}P than in the ^{45}Ca experiment. The present experiment confirms this view: Figure 3 shows that the rate of deposition of calcium in the tibial shafts can be calculated from the twenty-four hour activity values whereas the same calculation from the ^{32}P activity values (Fig. 4) does not yield a reliable value until forty-eight or seventy-two hours after the administration of the isotope.

After the administration of radiocalcium, the activity ratio of two parts of the skeleton gives the ratio of their respective rates of deposition of calcium (Bauer 1954*a*), provided a time interval is chosen (after the administration of isotope) which is long enough to preclude the factor of " exchange " and short enough to exclude the importance of resorption of the newly built-in bone salt. The activity ratio between the tibial shafts and the incisor teeth should thus be the same whether ^{45}Ca or ^{32}P values are recorded. Table I shows that the two sets of data agree.

TABLE I

ACTIVITY RATIOS TIBIAL SHAFTS/INCISORS AT VARYING INTERVALS AFTER THE SIMULTANEOUS ADMINISTRATION OF ^{45}CA AND ^{32}P TO YOUNG RATS

Interval after administration of the isotopes	^{45}Ca activity ratio tibial shaft/incisors	^{32}P activity ratio tibial shaft/incisors	Number of animals
48 hours	·40 (·040)	·43 (·010)	5
72 hours	·39 (·023)	·42 (·015)	5
96 hours	·38 (·046)	·39 (·031)	5
120 hours	·40 (·038)	·37 (·042)	5

Figures in brackets equal standard error of the mean.

The value of these ratios during the longer time intervals is about 0·4. Knowing that the deposition rate of calcium in the tibial shafts is 0·03 milligrams of calcium per hour (Fig. 3), the rate of deposition of calcium in the incisor teeth is thus 0·075 milligrams of calcium per hour. This figure is in agreement with earlier figures, in rats of the same strain and of about the same age, derived both from morphological methods and from isotope data (Bauer 1954*a*). From the above it is seen that the same holds true for the phosphorus values.

In an early isotope experiment (Armstrong and Barnum 1948) the conclusion was reached that there is a difference in the metabolism of the calcium and the metabolism of the phosphorus of the bone salt. It should be stressed, however, that the rate of deposition of bone salt can be determined only by referring skeletal activity to blood activity. This can be done either by direct reference (" U-values ") or indirectly by recording the ratios of two skeletal activity values which meet the requirements pointed out above. If these conditions are not met with, misinterpretations might arise.

SUMMARY

After the simultaneous administration of radiocalcium and radiophosphorus to young rats the rate of deposition of calcium and of phosphorus in various skeletal parts was computed. Agreement was found between the two sets of data. No difference was thus found in the metabolism of the calcium and of the phosphorus of the bone salt.

ADDENDUM

Since this paper was submitted for publication (December 1954) further analysis of *inter al.* the data above has revealed that the formation rate (accretion) of bone salt can be computed even *before* the " U-value " has reached a plateau. This method permits also a computation of the magnitude of the exchangeable Ca fraction. (Bauer, G. C. H., Carlsson, A., and Lindqvist, B. (1955): Evaluation of Accretion, Resorption, and Exchange Reactions in the Skeleton. Kongliga Fysiografiska Sällskapets i Lund Förhandlingar, **25,** Nr. 1.)

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THE CYSTS OF OSTEOARTHRITIS OF THE HIP

A Radiological and Pathological Study

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Bone cysts are a well recognised radiological feature of advanced osteoarthritis (Plewes 1940, Brailsford 1952). They are seen frequently in relation to the hip joint in both acetabulum and femur, but it is perhaps not always appreciated how numerous and extensive they may be. The present study attempts to correlate the radiological and pathological features of the cysts occurring in the head of the femur, and to elucidate their pathogenesis. A general account of the pathology of osteoarthritis of the femoral head, with a brief discussion of the cysts, was presented by Harrison, Schajowicz and Trueta (1953). The pathology of the cysts was described in detail by Landells (1953), who suggested that they were caused by the intrusion of synovial fluid under pressure. Landells believed that the cancellous trabeculae yield under the pressure transmitted through the fluid, and that the cysts are formed in enlarged marrow spaces. Accordingly, he interpreted the cysts as " virtually incomplete fractures " of the trabecular system, and attributed the development of dense bone around them to " bodily displacement " of trabeculae by fluid and to new bone formation in response to strain.

The hypothesis that subarticular cysts are excavations produced by synovial fluid is not new. It was propounded by Freund (1940) to explain the formation of cysts in rheumatoid arthritis and haemophilic arthrosis. In Freund's opinion synovial fluid would be forced into the subarticular bone through defects in the surface by the sustained rise in intra-articular pressure resulting from an effusion into the joint. But Cruikshank, Macleod and Shearer (1954) reported the development of cysts in rheumatoid arthritis without clinical evidence of a significant increase of synovial fluid in the affected joints.

In our opinion satisfactory proof that the defects in the bone in osteoarthritis are caused by synovial fluid has not been established. The evidence to be presented is believed to prove that such lesions are foci of bone necrosis caused by violent impact between the opposing surfaces of the joint in the absence of the protection of healthy articular cartilage. Such injury is not unlikely, and it would explain the development of " kissing " lesions, as described by Plewes (1940), on opposing surfaces of the joint. Our studies lend support to the view that cavities filled with synovial fluid are often, but by no means invariably, found in the bony defects. We differ from Landells, however, in believing that bone destruction precedes the entrance of synovial fluid, and suggest that the cavities filled with fluid are formed in the course of repair as the dead bone is removed.

The use of the word " cyst " is liable to give rise to confusion because radiologists and pathologists employ it in such different ways. To the radiologist a " cyst " is simply a circumscribed area of translucency in bone; to the pathologist the term connotes an encapsulation of gas or fluid in any tissue. Since many, but by no means all, of the " cysts " detected radiologically in osteoarthritis do contain encysted fluid, the two usages of the word " cyst " are clearly not synonymous. In the hope of avoiding confusion we shall restrict the word " cyst " to the radiological lesion and use such circumlocutions as " loculi of fluid," " encysted fluid," or simply " fluid " for a cyst in the pathological sense.

MATERIAL AND METHODS

The following observations are based on a study of the excised femoral heads of thirty-six patients with advanced osteoarthritis of the hip, treated by the method of Judet and Judet (1950). Thirty of the patients were suffering from primary osteoarthritis as defined by Mercer (1951), and in six the disease was superimposed on other lesions of the hip. All the patients were ambulant. They were selected for operative treatment because of pain both at rest and on movement. Most of them showed serious limitation of movement in the hip, but this was not an indication for operation in the absence of intractable pain. Operation was not advised merely because cysts had been detected radiologically.

Fourteen other femoral heads, excised in the treatment of fracture of the neck of the femur, have also been examined for the presence of cystic lesions. In ten the head was resected as a primary measure, and in the remaining four because the fracture showed no signs of union two or more months after injury; one femoral head had undergone avascular necrosis. The average age of the patients with a fracture of the femur was seventy-three years and of patients with osteoarthritis sixty years.

The excised femoral heads were radiographed in at least two planes at right angles. The severity of the osteoarthritic changes was graded in the manner suggested by Collins (1949) and all the cases were regarded as being Grade IV on the grounds of extensive loss of cartilage, osteophyte formation and eburnation, often accompanied by flattening of the articular surface. The specimen was then sawn into slices 0·5 to 1·0 centimetre thick and these were radiographed in series. In one case we injected a cyst with 40 per cent lipiodol before section and its extent and ramifications are shown in Figure 4. Histological examination was carried out in each case. The bone slices were fixed in 10 per cent formalin, decalcified and embedded in paraffin in the usual manner. Sections were cut at 10–20 micra and stained with haematoxylin and eosin. Specific features were investigated by Mallory's stain for connective tissue, Mayer's stain for mucin, and Weigert's fibrin stain, and Gomori's method was employed to detect haemosiderin. Serial sections were employed to a very limited extent.

RADIOLOGICAL OBSERVATIONS

In our experience cysts are detected pre-operatively in the hips of most, but not all, patients with advanced osteoarthritis. There appears to be considerable variation in the amount of cyst formation and in clinical radiographs it is often difficult to estimate with any exactness their number, extent and size. Most of the cysts in the femoral head are small and are situated close to the articular surface. Less common are the larger cysts which sometimes extend into the femoral neck (Fig. 1). We have found no correlation between the amount of cyst formation and the duration of the disease.

FIG. 1

Radiograph showing advanced osteoarthritis of the right hip. Extensive cyst formation in the femoral head and neck.

Radiographs of the excised but still intact femoral head provide little additional information about the cysts; occasionally they reveal cysts that had not previously been detected. On the other hand, radiography of the series of slices of the femoral head is an excellent method of examination. Typical examples of such radiographs are seen in Figures 2 (this femoral head is shown pre-operatively in Figure 1) and 6, which are representative of our series. By this method we have demonstrated cystic lesions in every patient with primary osteoarthritis.

Small subarticular cysts, many too small to be detected pre-operatively, are surprisingly common, while larger cysts—some of unsuspected magnitude—are more clearly visualised.

In the six patients with secondary osteoarthritis, small cysts were seen in each femoral head although none had been detected in radiographs taken before operation. No cyst was demonstrated either before or after operation in the patients with fractures of the neck of the femur.

CORRELATION OF THE RADIOLOGICAL AND MACROSCOPICAL APPEARANCES

In advanced osteoarthritis the femoral head is characteristically deformed (Harrison, Schajowicz and Trueta 1953) (Fig. 2, section 5). Most of the central part of the articular surface is denuded of cartilage and the cysts occur beneath the exposed bone (Figs. 2 to 7). We have not encountered a cyst beneath intact articular cartilage, and only rarely in the infero-medial osteophyte. The surface bone overlying the cysts is eburnated and sclerosed, particularly on the superior aspect of the head. It is often stippled with minute pits, most

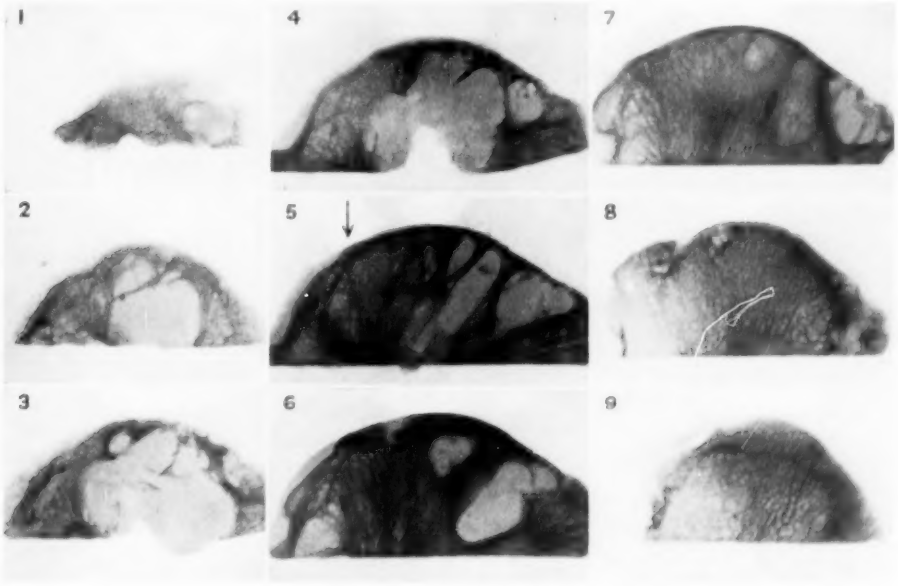

FIG. 2

Radiograph of the series of bone slices of the femoral head illustrated in Figures 1 and 3, showing the situation and extent of cyst formation. The arrow indicates the original outline of the femoral head now covered by a large osteophyte.

of which end blindly just below the surface, although a few perforate the superficial bone overlying a radiological lesion. Sometimes there are small islands of cartilage in the denuded area, and on section this cartilage is seen to plug a defect in the surface bone, commonly overlying a larger defect in the underlying trabeculae. These fragments of cartilage are sometimes assumed to be a survival of the original layer, but histological examination clearly shows that the great majority are formed by metaplasia during repair of local damage to the bone.

The macroscopic appearance of the cysts shows great variation. Most are small, roughly spherical defects in the sclerosed subarticular bone (Fig. 2, section 4). The larger lesions are

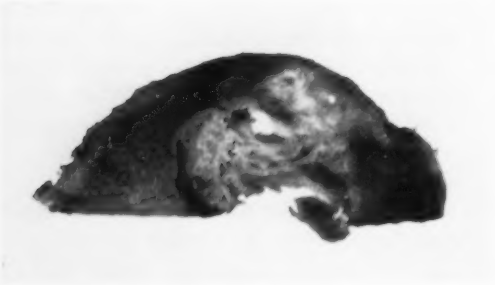

FIG. 3

A large irregular cyst in cross-section. It is covered by a thin layer of denuded articular bone. The radiograph of this lesion is shown in Figure 2, section 4. Note that small subarticular cysts are more clearly visualised by radiography.

less often spherical (Figs. 3 to 7) and may pursue an oblique course towards the articular surface. Even when there is such extensive destruction of the trabecular system as is shown in Figures 2 to 7 a thin plate of bone is usually present at the overlying surface. Histological examination frequently shows that this bone is in fact necrotic but we have been unable to identify necrotic bone in radiographs of the slices.

The defect in the trabecular system is occupied by connective tissue which usually, but by no means always, contains one or more loculi (Figs. 3 to 7) filled with thick, mucoid fluid that is occasionally bloodstained. Usually the greater part of the bony defect is occupied by such loculi, but in our experience there is no correlation between the extent of the defect in the bone and the size of the encysted loculi of fluid. Many loculi of fluid are spherical or

FIG. 4

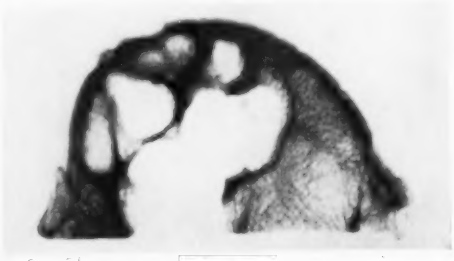

FIG. 6

FIG. 5

FIG. 7

Figure 4—Radiograph of excised femoral head to show the extent of a large cyst. Lipiodol has been injected (see text). Figure 5—The same cyst in cross-section. Figure 6—Radiograph of the section showing the extent of the bone defect. Figure 7—Photomicrograph of the same cyst showing scar tissue and loculi in the bone defect. New formation of cartilage (opposite arrows) has filled defects at the overlying surface resulting from bone destruction. (H. and E., × 2.)

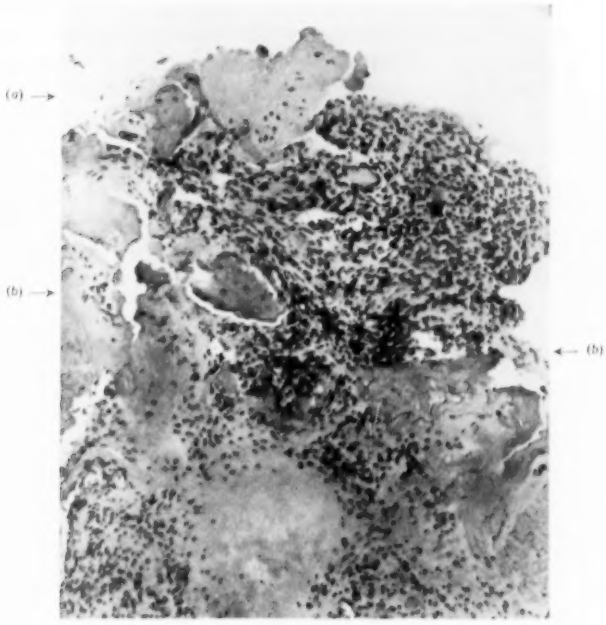

FIG. 8

A focus of recent damage at an early stage of repair. Note the disrupted cartilage and bone at the surface (*a*) and the fractured trabeculae in the deeper part of the lesion (*b*). The lesion is filled with a highly cellular granulation tissue similar to that produced at the site of fracture. (H. and E., ×85.)

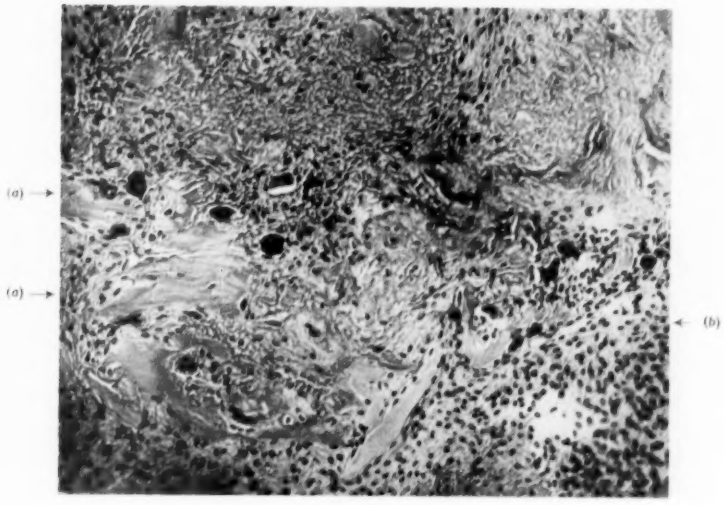

FIG. 9

The same focus as in Figure 8 but at a different level. Note the presence of large, darkly staining osteoclasts in relation to the spicules of fragmented bone (*a*) and the proliferation of undifferentiated cells (*b*) throughout the site of damage. (H. and E., ×130.)

pyriform, as Landells observed, but some conform with the irregular shape of the defect in the bone (Figs. 3 to 7). Some are incompletely partitioned by septa of connective tissue. Occasionally they communicate with the joint cavity through the minute defects observed in the articular surface, but in our experience such communications are rarely found, and the loculi are usually separated from the joint cavity by a thin plate of bone (Fig. 3) or by cartilage occupying defects in the surface plate.

HISTOLOGICAL OBSERVATIONS

In the following account our object is to review the nature of the damage to the bone at the sites of cyst formation and to describe the process of repair and the formation of cavities of fluid in the bony defects.

At the site of recent injury to the articular surface the surviving cartilage is disrupted, the subchondral plate is destroyed, and fractures are produced in the subjacent trabeculae

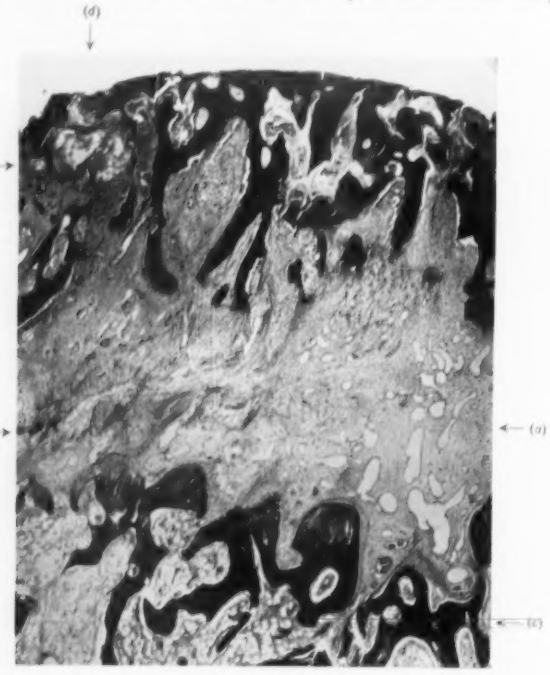

FIG. 10

Part of a large "cyst" which involves most of the femoral head. There is extensive loss of trabeculae (*a*) deep below the surface with replacement by scar tissue. The sclerotic subarticular bone shows extensive necrosis. Small loculi of fluid (*b*) are only present beneath a small defect (*d*) in the surface plate. The bone in the deeper part of the femoral head (*c*) is not necrotic. (H. and E., × 15.)

(Figs. 8 and 9). Usually, however, such foci of injury are at a more advanced stage of repair by the time the femoral head is excised. Nevertheless the changes that result from repair can usually be interpreted and the extent and character of the original damage assessed from the persisting remnants of necrotic bone, both at and below the surface, and from the disappearance of a segment of the original trabecular system (Figs. 12 and 13).

In the later stages of the disease, when the more superficial bone is sclerosed, injury is likely to produce necrosis rather than fragmentation of this surface bone. The force of impact

would be transmitted to the weaker underlying bone with resultant disruption and necrosis. This may result in loss of part of the trabecular system beneath an unbroken surface (Figs. 10 and 11). We have been impressed by the similarity of such lesions to those circumscribed defects that contain encysted fluid, where, in our experience, it is also usual to find necrotic bone at or below the surface (Figs. 14 to 16). Arterial thrombosis is sometimes seen within the necrotic segment and may be partly responsible for the development of such necrosis. However, we have seldom found thrombosed arteries proximal to the site of injury, and arteriosclerosis is never severe enough to seem a probable cause of serious ischaemia.

Repair of this bone damage bears a striking resemblance to the changes seen at the site of an imperfectly immobilised fracture. We have not seen a necrotic focus before the onset of repair, presumably because a period of comparative inactivity preceded operative removal of the femoral head. In a focus of recent injury (Figs. 8 and 9) the fragments of disrupted bone are being removed by osteoclasts, and a highly cellular and vascular granulation tissue,

FIG. 11

Part of the section shown in the top right hand corner of Figure 10. The bone and marrow (*a*) are necrotic and fractures have developed in the trabeculae (*b*). Note the absence of fragmentation of the surface. (H. and E., × 85.)

histologically similar to that seen at the site of fracture, fills the damaged focus and extends to the overlying surface. From an examination of many lesions at various stages of repair, we have concluded that cells with the potential capacity to form new bone, cartilage, or simply connective tissue are usually formed from the ends of interrupted trabeculae and surface plate, whereas granulation and scar tissue are usually formed from the bone marrow. Most foci of repair contain tissue derived from both sources (Fig. 12), but the relative amount varies in different lesions. Seams of osteoid may develop in the reparative tissue and so give

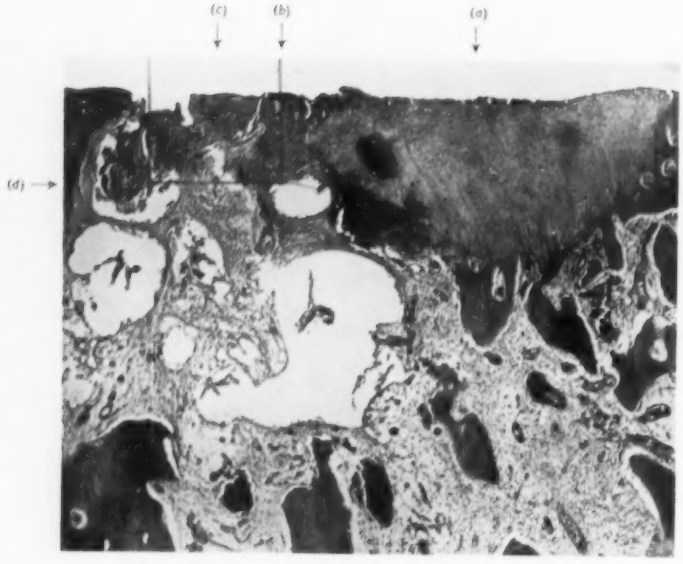

FIG. 12

Healing of a focus of damage. The surface is repaired by cartilage (*a*) arising from fractured trabeculae. Living bone is present at (*b*) and fractured dead bone at (*c*). The central zone of greatest bone destruction is filled with oedematous connective tissue derived from the marrow spaces, and contains several loculi of fluid. Note the presence of bone debris in the loculus (*d*) immediately below necrotic surface bone. (H. and E., ×30.)

FIG. 13

Area outlined in Figure 12 to show the presence of fractured necrotic bone at the surface (*a*); living bone at (*b*). (H. and E., ×160.)

FIG. 14

A large irregular bone defect containing several loculi of fluid (*a*). The overlying surface is extensively damaged; a persisting defect is present at (*b*); new cartilage has replaced the surface plate at (*c*). (H. and E., × 5.)

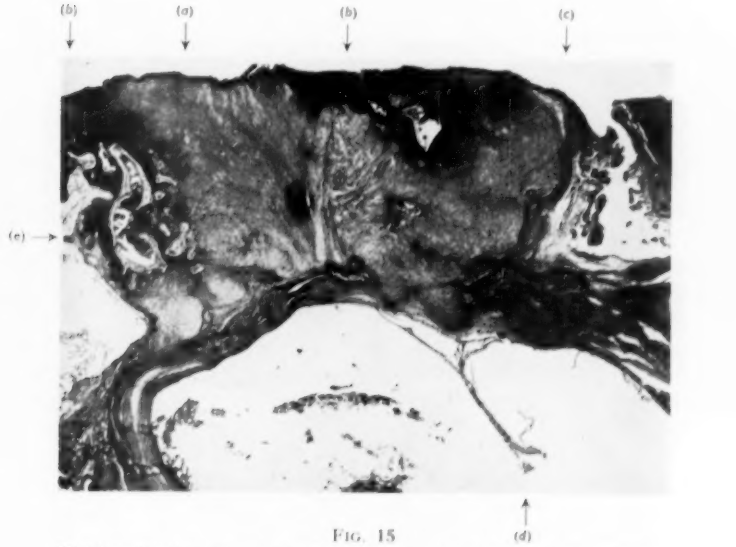

FIG. 15

Field outlined in Figure 14. Fibrocartilage (*a*), derived from surviving fragments of the surface bone (*b*) occupies most of the breach in the surface. A defect (*c*) that may connect with the underlying loculi of fluid (*d*) is still present. Small loculi are seen at (*e*). (H. and E., × 5.)

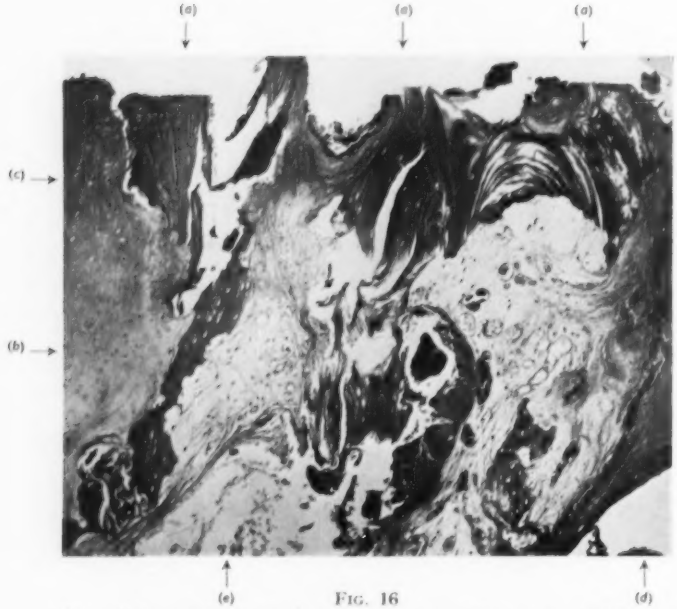

FIG. 16

Necrotic bone (*a*) at the surface of the large cyst illustrated at a different level in Figs. 14 and 15. The loculi of fluid (*d*) and (*e*) in Figure 15 are seen at (*d*) and (*e*). Some of the dead bone has a "moth-eaten" appearance which is probably due to osteoclastic activity, and is surrounded by vascular, oedematous connective tissue. Cartilage (*b*) has formed from interrupted living bone (*c*) at the surface. (H. and E., ×65.)

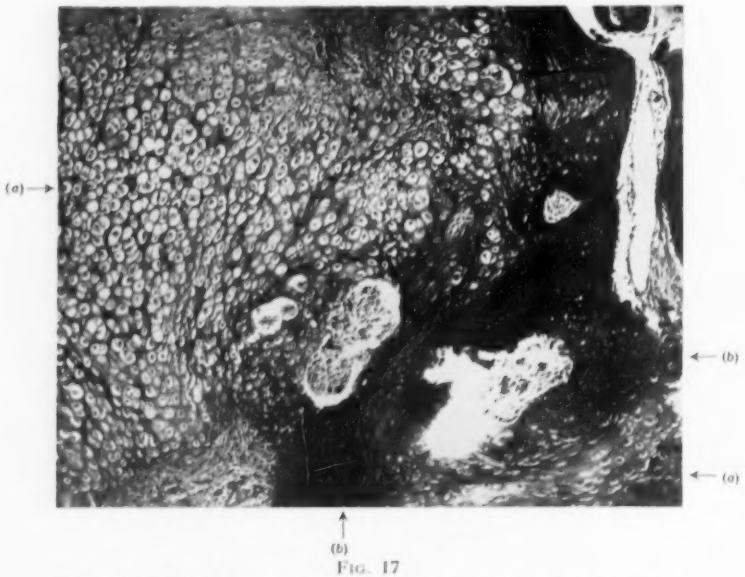

FIG. 17

Hyaline cartilage (*a*) occupying defects in the trabecular system. Note that the cartilage is directly continuous with interrupted trabeculae (*b*). (H. and E., ×65.)

rise to new trabeculae (Fig. 18) or restore the damaged surface. The new bone is readily identified by its unusual cellularity and by its abnormal internal architecture. Instead of new bone, masses of cartilage may form by a process of metaplasia (Figs. 7, and 12 to 17) and some is converted into scar tissue (Fig. 10). As these illustrations show, the origin of cartilage or connective tissue from injured bone may be inferred when these tissues are in direct continuity with interrupted trabeculae. We believe that the cartilage found at the eburnated surface is formed in this way, and is rarely a surviving fragment of the original articular layer. When healing is complete, new bone, cartilage or connective tissue separate the persisting defect in the trabecular system from the synovial cavity.

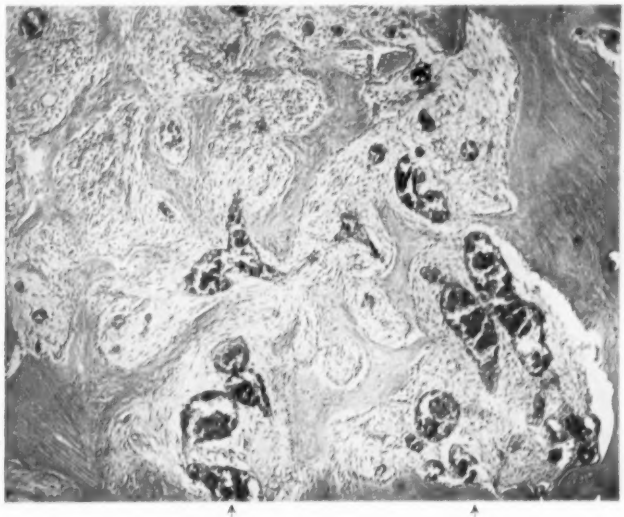

FIG. 18

Newly formed seams of trabecular bone in a wedge-shaped defect which extends to the surface. The new bone is in continuity with the original trabeculae (*a*). Note engorged sinusoidal vessels (*b*) in the area of repair. (H. and E., ×65.)

When dead bone persists in the foci of damage, it is enclosed in scar tissue, or incarcerated in newly formed bone or cartilage. The necrotic surface plate is slowly replaced in some instances by new bone growing in from the margin of the lesion. The amount of osteoclastic activity in relation to the dead bone is extremely variable, and in many long-standing lesions there is little evidence of continued bone removal (Figs. 11 and 16).

There is strong circumstantial evidence that synovial fluid enters the bony defects through a breach in the articular surface (Landells). We have confirmed that a communicating channel is sometimes present between the encysted fluid and the synovial cavity (Figs. 10 and 12) but as we did not study this point by serial sections, we feel that many were probably missed, although others may well be obliterated as the surface is repaired. On the other hand, we have been impressed by our failure to find such loculi when a defect in the trabecular system is covered by an intact surface (Fig. 10), and also at sites of recent injury (Figs. 8 and 9).

The loculi of fluid are surrounded by connective tissue of very varied appearance. In comparatively recent lesions the connective tissue is loose and oedematous (Fig. 12), and contains fibroblasts and capillaries, histiocytes swollen with debris, and lymphocytes. In older lesions more mature connective tissue is present (Fig. 7); it is often condensed around the loculi to form a capsule, and contains many large sinusoidal blood vessels (Harrison, Schajowicz and Trueta 1953) presumably derived from the capillaries formed during repair. In some

lesions the scar tissue is extremely dense, particularly around the loculi of fluid (Fig. 14). In some loculi, strands of necrotic tissue stretch across the cavity and are attached either to the capsule or to the septa from which they appear to originate (Figs. 14 and 15). Traumatic haemorrhages, often associated with thrombosis of small vessels, are common, particularly near the surface of the cysts, and bleeding may consequently occur into the loculi of fluid. However, we found no evidence that haemorrhage plays any part in the production of the cysts (Pommer 1913, Lang 1932, 1934).

We have never seen " bodily displacement " (Landells) of trabeculae around encysted fluid, even at an early stage of repair, and orientation of the trabeculae is not present at this stage (Fig. 12). Around the older cysts however the trabeculae do undergo remodelling and sclerosis (Fig. 14) and new bone sometimes continues to form slowly from seams of osteoid in the marginal connective tissue. This surrounding sclerosis is probably caused partly by pressure transmitted through the encysted fluid, but it may well be further increased by the stress thrown on these marginal trabeculae by the loss of a segment of bone from the weight-bearing area.

DISCUSSION

It is the very nature of osteoarthritis to expose the subarticular bone to injury, particularly in the weight-bearing joints. In our histological studies we have emphasised the importance of such injury as the cause of cyst formation during the evolution of the disease.

It is our belief that the shock of jolting movements at the hip is normally absorbed by the protective resilience of healthy articular cartilage and that in osteoarthritis such movements injure the bone at the site of impact and the shock is transmitted to the subjacent trabeculae. The nature and extent of the damage to the bone will depend on the degree of protection afforded by any surviving cartilage, the presence and extent of sclerosis in the subarticular bone and on the severity of the injury. Injury to the delicate subarticular bone before the cartilage is completely lost results, we believe, in the numerous small foci of bone destruction that we have described in the vicinity of the articular surface; the surface plate and the subjacent trabeculae are fractured and even fragmented, and the defect is open to the synovial cavity. At the opposite extreme, in the later stages of osteoarthritis, jolting injuries will affect densely sclerotic bone. In our experience injury at this stage is more likely to produce necrosis with less disruption of the superficial bone at the site of collision; at the same time, however, transmission of the force of impact through the trabecular system is liable to result in fractures and necrosis of weaker trabeculae, deep below the surface.

The presence of cavities filled with fluid at the site of injury is certainly the result of disruption of the articular surface so that synovial fluid is forced into the bone under the pressure of the weight of the body. We agree that the appearance of large defects filled with synovial fluid might reasonably be interpreted in the way suggested by Landells, but an examination of early lesions at different stages of evolution fails to provide evidence that bone destruction or " bodily displacement " of trabeculae is actually caused by an influx of synovial fluid. As we have shown, synovial fluid is not always present at a site of recent injury, even when the surface bone is disrupted, and fluid is absent from sites of bone necrosis when the continuity of the surface is not destroyed. We believe that such observations provide evidence that the bone destruction precedes, and is independent of, the entrance of synovial fluid into the bone, and that if the surface remains intact until the deranged focus is filled with granulation tissue the formation of cavities of synovial fluid will not occur. On the other hand, should synovial fluid enter the necrotic bone, it is likely to disrupt the already devitalised tissue, which may then be swept into the cavity of the joint, leaving a potential space in which synovial fluid becomes encysted during the stage of repair.

Extensive necrosis of the femoral head in osteoarthritis was interpreted by Pridie (1952) as the result of a " vascular catastrophe " and was aptly likened by Collins (1939) to the histological picture of avascular necrosis. The association of cystic lesions with such bone

necrosis was reported by Harrison, Schajowicz and Trueta (1953) who believed that a " series of infarctions " occurs in the " destructive phase " of osteoarthritis. These authors believed that infarction of the bone was caused by " collapse " of the femoral head, and concluded that " ischaemic changes " were a usual occurrence in the late stages of the disease. We agree that ischaemia may play a role in the development of these foci of bone necrosis, but in our opinion it is the result of arterial damage sustained at the time the bone is injured, and collapse of the femoral head is more likely to be the result of extensive bone destruction than its cause.

These sites of fracture and necrosis may become visible radiologically once devitalised fragments of bone have been removed, either by osteoclastic activity or by being shed into the cavity of the joint. In many instances the radiographic defect is perpetuated by the production of cartilage and connective tissue instead of new bone, and by the formation of cavities of synovial fluid. Such imperfect healing, analogous to that at the site of an imperfectly immobilised fracture is, we believe, also caused by lack of rest. Continued use of an injured osteoarthritic hip must result in the repeated transmission of the weight of the body through the defect in the trabecular system. In consequence, healing is likely to be impaired and the cavities of synovial fluid will tend to persist instead of being obliterated by granulation tissue. It is our contention, therefore, that the cysts of osteoarthritis are sites of injury that often, if not invariably, contain encysted synovial fluid, and the persistence of the radiographic defect is an indication that continued use of the joint has impeded the natural processes of repair.

SUMMARY

1. An attempt has been made to correlate the radiographic appearances and the morbid anatomy of the cystic changes that occur in the head of the femur in advanced osteoarthritis.
2. The suggestion is made that these lesions are foci of traumatic bone necrosis. Repair may be complicated by the subsequent entrance of synovial fluid through defects in the surface.

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THE SIGNIFICANCE OF METALLIC TRANSFER IN ORTHOPAEDIC SURGERY

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It is common practice in modern orthopaedic surgery to insert pieces of metal into the human body; often they remain in the tissues for considerable periods. It is clear that in addition to having the necessary mechanical properties to ensure adequate immobilisation of the bone these metals should be inert in tissues and interfere as little as possible with repair and normal physiology. All appliances inserted by orthopaedic surgeons are made of metal having a very high corrosion resistance: during an operation, however, they are handled with tools which are not made of the same material. The behaviour of metals immersed in fluid depends markedly on the specific nature of their surfaces, and it is interesting, therefore, to consider what effect this handling has on the metal surfaces and whether it could cause any change in the reaction of the buried metal with the body tissues. It is first necessary to appreciate what the surface of a metal is like, and what happens when it is touched by another solid.

THE NATURE OF THE CONTACT BETWEEN METALS

Metal surfaces, no matter how carefully they are prepared, are never flat. They have irregularities which are many atoms high; even using the most advanced polishing techniques it is impossible to create atomically smooth surfaces. Whenever two metals are brought together contact first occurs at the points where the tips of the asperities on one meet the surface of the other. Initially the force driving the metals together is transmitted through these small areas of contact only, and consequently the local pressure is very great; so great in fact that it exceeds the yield pressure of the metals and plastic deformation occurs. The region of contact grows as the two surfaces deform to accommodate each other until it reaches such a size that the mean pressure over the actual area of contact no longer exceeds the yield pressure of the metal. Thus, although two metals may apparently be in contact over several square centimetres, in fact they touch only at a few isolated points. The total area of true contact may be as little as one-millionth of the apparent area.

Over this small region, however, the metals are very strongly welded together. The local deformation of the metal which occurs near the areas of contact makes these regions stronger than the bulk of the solid. Consequently when the metals are separated they often part not at the original interface but slightly to one side of it, in the region which has not been strengthened by deformation. This means that small fragments of one metal are left behind on the surface of the other, and remain adhering firmly to it. It is almost impossible to touch two metals together without causing some transfer of this kind. The amount of metal transferred is greatly increased if any slip occurs between the metals and also depends markedly on the presence or absence of lubricating and other films on the surfaces. Figures 1 and 2 show two photomicrographs illustrating typical fragments of copper welded on to steel after the copper had been slid once over the steel surface.

A general account of the researches on which these conclusions are based was given by Bowden and Tabor in 1950.

In simple mechanical operations in which a tool is brought into contact with a metal some transfer may be expected to occur from one to the other. In orthopaedic surgery, for example, we may expect that tiny particles of the screwdrivers, spanners and forceps used in an operation may remain welded to the screws, bolts, and plates which are buried in the

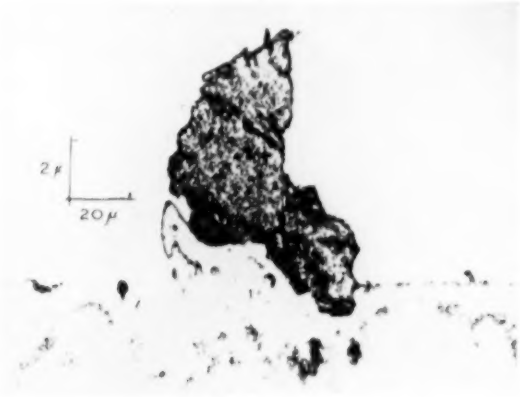


FIG. 1

Photomicrograph of section of steel surface after a piece of copper had been slid over it. A fragment of copper (dark) is seen adhering to the steel surface. (Dr A. J. W. Moore.)

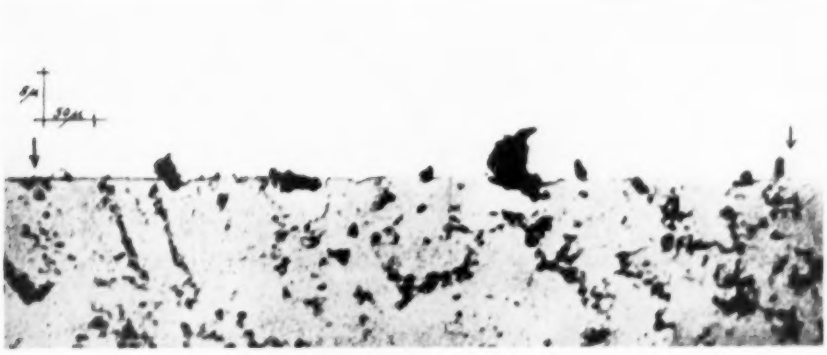


FIG. 2

Section of steel surface showing fragments of copper adhering to it and rupture of the steel surface. The track width is marked by arrows. (Dr A. J. W. Moore.)

body. If fragments of metal from the tools do become buried in the patient in this way they could act as corrosion centres and initiate a continuing dissolution of the buried metal.

Experiments have been carried out to investigate whether such metallic transfer does occur, and to study its amount and distribution. A brief account of some of our earlier experiments has been given elsewhere (Bowden, Williamson and Laing 1954*a*).

EXPERIMENTAL METHODS

Three procedures common in bone surgery have been studied: inserting a screw into a bone, drilling a pilot hole for it, and tightening a bolt in its socket. The screws, bolts, plates and all the tools used were samples of those supplied to the medical profession. Their specifications are given in Table I.

TABLE I

SPECIFICATIONS OF MATERIALS

Element	Per cent by weight							
	(*a*)	(*b*)	(*c*)	(*d*)	(*e*)	(*f*)	(*g*)	(*h*)
Carbon	0·07	trace	0·12	0·70–0·80	0·30	0·16	0·19	0·08–0·15
Chromium	18·0	30·0	18·0	4·0–4·25	13·0	16·50	13·60	17·0–20·0
Nickel	8·0	—	8·50	—	—	2·50	0·34	7·0–10·0
Manganese	0·50	trace	0·80	—	0·30	0·20	0·20	2·0 max.
Silicon	0·30	trace	0·60	—	0·35	0·20	0·24	0·75 max.
Molybdenum	2·75	3·0	—	0·80 max.	—	—	—	—
Phosphorus	—	—	—	—	—	—	—	0·03 max.
Sulphur	—	—	—	—	—	—	—	0·03 max.
Cobalt	—	65·0	—	—	—	—	—	—
Tungsten	—	—	—	18·0	—	—	—	—
Vanadium	—	—	—	1·0–1·30	—	—	—	—

(*a*) Firth Vickers F.M.B. steel: used for plates, screws, etc.
(*b*) Vitallium: used for plates, screws, etc.
(*c*) Firth Vickers F.S.T. steel: used for stainless steel twist-drills.
(*d*) High speed steel: used for engineers' twist-drills.
(*e*) Firth Vickers F.H. steel: used for circular saw.
(*f*) Firth Vickers S80 steel: used for box-spanner for Vitallium bolts.
(*g*) Martinsitic steel used in all other tools.
(*h*) Fink's recommendation (AISI 302); current American specification (18 8 S Mo or AISI 316–317) quotes 10–14 per cent nickel, 0·08 per cent max. carbon, and 2–4 per cent molybdenum.

In each operation studied the head of the tool used was made radioactive by irradiating it in the neutron pile at Harwell. It was then used in a manner simulating as closely as possible the conditions of an actual orthopaedic operation and the amount of metal transferred from it was measured using either a Geiger counter or a scintillation counter. The specific activity of the tool under investigation was determined by counting the emission of a known mass of it, and hence the mass of transferred metal could be calculated.

The distribution of the metallic transfer was determined by an autoradiographic technique. In this method radioactive particles are made to record themselves by their own radiation on a sheet of sensitive film placed against the contaminated surface.

RESULTS

In the first series of experiments stainless steel screws were driven into bone with a steel screwdriver. The mass of screwdriver metal transferred to the screw heads was found to depend markedly on the amount of slip that occurred between the driver and the screw. Table II shows the values found in four typical cases, together with the results of an analogous experiment in which the same screwdriver was used to insert a series of Vitallium screws.

TABLE II

TRANSFER FROM STEEL (*g*) SCREWDRIVER TO STAINLESS STEEL (*a*) SCREWS AND TO VITALLIUM (*b*) SCREWS

	Steel screws	Vitallium screws
Screw 1. No detectable slip	17 μg	0·6 μg
Screw 2. Slight slip	22 μg	0·8 μg
Screw 3. Some slip	45 μg	1·0 μg
Screw 4. Much slip	65 μg	1·7 μg

The values quoted in Table II were measured to an accuracy of $\pm$10 per cent, except the two values below 1 μg which are accurate to $\pm$20 per cent.

TABLE III

TRANSFER FROM STEEL (*g*) SPANNER TO STAINLESS STEEL (*a*) BOLTS AND FROM STEEL (*f*) SPANNER TO VITALLIUM (*b*) BOLTS

	Steel bolts	Vitallium bolts
Bolt 1. Spanner applied once only. No detectable slip. Great force used	38 μg	3·6 μg
Bolt 2. Spanner applied twice. Some slip. Normal force used . .	43 μg	6·4 μg
Bolt 3. Spanner applied six times. Much slip. Very great force used	103 μg	20 μg

In the second series of experiments the transfer occurring during bolting was studied. Hexagonal-headed bolts were gripped by their shanks in a vice and a radioactive surgical box-spanner was used to tighten them aginst the grip of the jaws. Forces similar to those used in a surgical operation were applied. The spanner was removed and replaced several

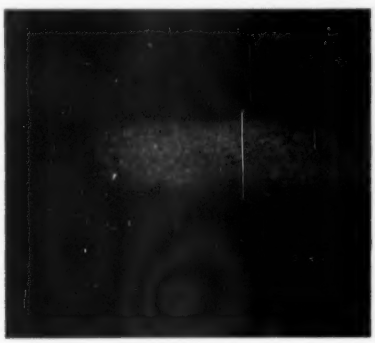


FIG. 3

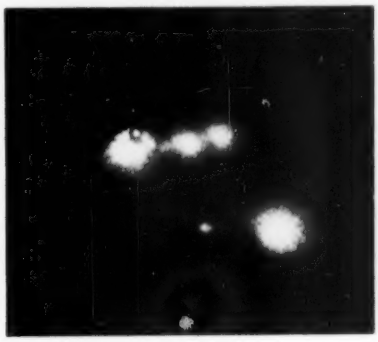


FIG. 4

Autoradiographs of transferred metal on stainless steel screw heads. Figure 3—No detectable slip of screwdriver, 17 μg. Figure 4—Some slip of screwdriver, 47 μg.

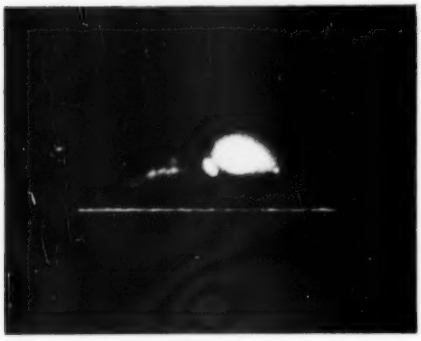


FIG. 5

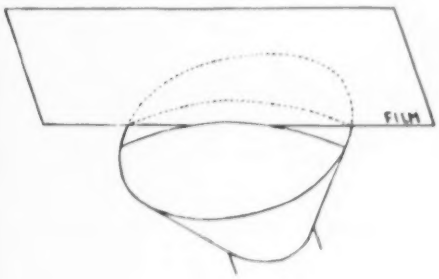
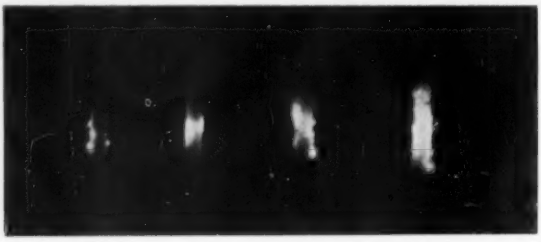


FIG. 6

Figure 5—Autoradiograph of transferred metal within the slot of a stainless steel screw. The white line represents the edge of the film and the bottom of the slot. (Dr A. J. W. Moore.) Figure 6—Position of film for autoradiograph.

times as repeated application is customary in orthopaedic surgery. Table III gives the transfer observed to stainless steel bolts, and also to Vitallium bolts which were investigated in a similar experiment. Again the transfer was measured to an accuracy of ± 10 per cent.

Figures 3 and 4 show typical autoradiographs of the transferred metal adhering to screw heads. These were obtained by standing the screws head downwards on a photographic film. Both autoradiographs show a grey region caused by radiation emanating from transferred metal within the slot of the screw, and several small areas of intense whitening indicating discrete transferred particles are visible along the outer edges of the slot of the screw on which



(*a*) (*b*) (*c*) (*d*)

FIG. 7

Autoradiographs of transferred metal on Vitallium screws. First screw—No slip, 0·6 μg. Second screw—Slight slip, 0·8 μg. Third screw—Some slip, 1·0 μg. Fourth screw—Much slip, 1·7 μg.

the driver had slipped. The distribution of transferred metal within the slot was obtained by inserting the edge of a sheet of film into it. The method used and the autoradiograph obtained are illustrated in Figures 5 and 6. Figure 7 is an autoradiograph of the heads of four Vitallium screws. It shows clearly the increase in transfer which occurs when the driver is allowed to slip in the screw slot. From the information displayed in Figures 3 to 7 it is deduced that the metal transferred from the driver is distributed on the screws as shown in Figure 8. Similar autoradiographs of bolts which have been tightened by a box-spanner support the view that transfer is greatest in those regions where most slip occurs.

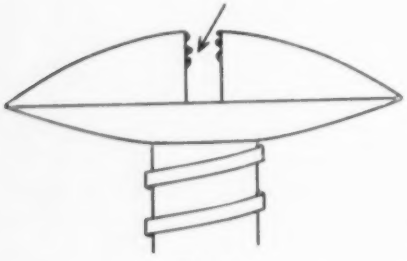


FIG. 8

Distribution of transferred metal on screw head.

It is usual to prepare a pilot hole for an orthopaedic screw, using either an electric or hand-operated drill. Both engineers' twist-drills and special stainless steel ones are commonly used for this purpose. Frequently pilot holes are drilled after a plate has already been fixed in place by two or more screws, the remaining holes of the plate being used to localise the twist-drill. Owing to the convex and wet surface of the bone it sometimes happens that the twist-drill comes into contact with the edges of the plate hole, and considerable friction may occur between them. Figure 9 shows a typical hole in an orthopaedic plate which was removed because of persistent sepsis after being attached to the right tibia of a patient for five months. It is clear that the twist-drill has ground away the shoulder of the hole.

In collaboration with Colonel J. Hickman, of the Department of Veterinary Clinical Studies, Cambridge, experiments have been carried out in which the transfer occurring from twist-drills to bone and to a plate have been measured during *in vivo* experiments on an eighteen-month old Collie-cross bitch. Holes were drilled through one cortex of the femur with a hand-operated drill. The holes were sectioned axially and both halves assayed together. It was found that the amount of metal transferred to live bone when a pilot hole is drilled

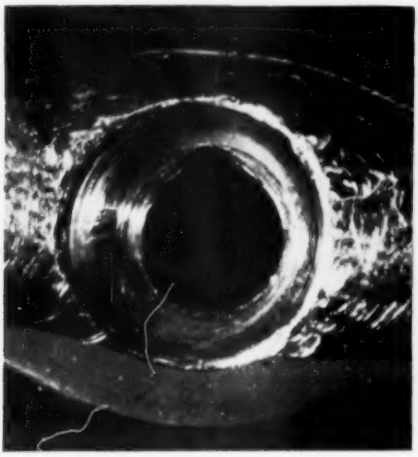


FIG. 9

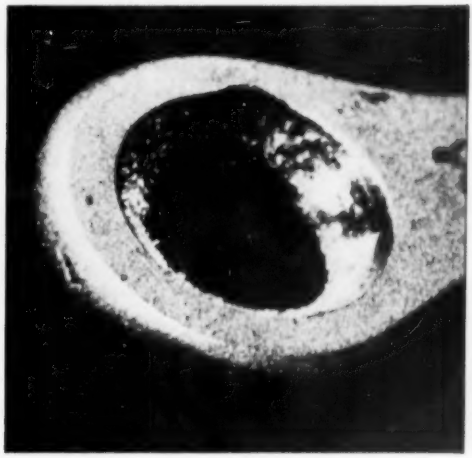


FIG. 10

Figure 9—A typical hole in an orthopaedic plate. This plate was removed from the right tibia of a patient. Note that the twist-drill has ground away the side of the hole. Figure 10—The side of this plate hole was ground away with a radioactive twist-drill until the damage appeared similar to that occasionally observed in orthopaedic plates removed from the body. More than a milligram of metal from the tool was transferred to this plate.

decreases markedly with the age of the twist-drill used. The transfer to the surface surrounding the first hole made by a $\frac{7}{64}$-inch stainless steel drill was 12 μ gramme; this decreased to 5 μ gramme with the second hole and none was detected with subsequent holes. Similar behaviour was observed using engineers' drills, the corresponding amounts being 0·9 μ gramme and 0·5 μ gramme. This behaviour agrees closely with that reported by Shaw using human bones thawed from a bone bank (Bowden, Williamson and Laing 1954c).

The transfer of metal to a plate when a pilot hole was drilled through it was, however, very much greater. In the first experiment the drills were merely allowed to touch the edges

TABLE IV

TRANSFER TO PLATES FROM DRILLS WHEN A PILOT HOLE IS DRILLED THROUGH A PLATE HOLE

	Drill allowed to touch plate occasionally		Drill allowed to grind against plate	
	Engineers' drill (*d*), V.H.N. 860	Stainless steel (*c*) drill, V.H.N. 400	Engineers' drill (*d*), V.H.N. 860	Stainless steel (*c*) drill, V.H.N. 400
Stainless steel (*a*) plates, V.H.N. 300–350 . .	2·3 μ grammes	17·5 μ grammes	3·0 μ grammes	1,500 μ grammes
Vitallium (*b*) plates, V.H.N. 400. . . .	0·55 μ grammes	1·75 μ grammes	1·2 μ grammes	2,000 μ grammes

of the plates during the drilling process. In the second the shoulders of the plate holes were deliberately ground away with the drill until the damage appeared similar to that occasionally observed in plates removed from patients (Fig. 10). Table IV shows the amounts measured when both stainless steel and engineers' drills were used to make pilot holes through stainless steel and Vitallium plates.

The large amounts of transfer observed with stainless steel twist-drills are probably due to the fact that the plates are as hard as these drills and bulk shearing of the cutting edge of the drills occurs.

In further *in vivo* experiments the amount of metal left adhering to the surface of a bone graft was investigated. A radioactive stainless steel circular saw was used to cut a graft from a canine tibia, and it was found that 320 μ grammes of the saw became embedded per square inch of the cut surface. Other experiments investigating the amount of metal transferred during routine engineering operations suggest that appreciable transfer may occur when surgical mallets are used to insert orthopaedic nails.

THE CHOICE OF A METAL FOR ORTHOPAEDIC SURGERY

Before 1938 a wide variety of metals was used by orthopaedic surgeons for the internal fixation of fractures and similar purposes. In the opinion of Venable and Stuck (1938) these metals failed because they caused a reaction in the surrounding tissues from the electro-chemical corrosion of the buried metal. The presence of these corrosion currents is believed to affect the growing tissue around a fractured bone or at an operation site and to cause the phenomenon known as electrolytic inflammation. It is not known definitely whether it is the presence of the currents themselves or the increased concentration of metal ions in the tissue which they produce that causes electrolytic inflammation. However, the condition interferes with the union of the fracture and also produces heavy fibrosis around the plate or nail, occasionally leading to the rejection of the metal. If two different metals are left in the same patient the electrolytic action can be so great that one of them may be dissolved in bulk.

In an attempt to remove the danger of electro-chemical corrosion it was decided in America to standardise a single metal for the manufacture of all internal appliances. Between 1941 and 1948 investigations were carried out to find the most suitable metal for use in orthopaedic surgery (Fink and Smatko 1948, Venable and Stuck 1947, 1948). Two main factors were considered in this work: firstly, the ideal metal has to be inert when buried in the tissues; and secondly, it must possess the necessary mechanical strength to withstand the stresses to which it might be subjected.

Of the metals investigated AISI 302 stainless steel and Vitallium were found to be the most inert, Vitallium being slightly the more so (Fink and Smatko 1948). The specifications of these metals are given in Table I. Stainless steel was felt to have certain advantages over Vitallium because of its more suitable physical properties. Not all surgeons were in agreement over this and both metals have come into wide use. It was, however, recognised that all the screws and plates used in any one case should be of the same composition in order to avoid electro-chemical corrosion, and it was recommended that stainless steel material should be damaged as little as possible during insertion to avoid increasing its toxicity to the tissues, and that the drills used should also be made from the same steel as the plates and screws. From the point of view of cheapness and availability stainless steel presents advantages over Vitallium and similar alloys. This is especially true when the surgeon needs a new or modified appliance, because this can more easily be made from stainless steel than from Vitallium; the latter requires casting and a complicated checking system to guard against physical defects in the appliance. A survey of current practice in orthopaedic surgery was given by Watson-Jones (1954).

The screws, bolts and plates manufactured by Down Bros. and Mayer & Phelps are made from Firth Vickers F.M.B. steel, which is within Fink's specification, and these products were used in our tests. The manufacturers have attempted to increase the inertness of their F.M.B. stainless steel products by reverse electrolytic plating (to remove surface metallic debris) and by passivation in nitric acid (to increase the thickness of the protective surface layer of oxide). The tools used were manufactured from a martinsitic steel (Table I).

While it is unusual nowadays to find bulk corrosion of buried stainless steel components, over the course of the last few years it has been observed that the tissue reaction to the

so-called non-toxic stainless steel varies from case to case. This variation has also been seen from place to place around the same internal appliance. Something appears to be making the "non-toxic" metal "toxic" to the tissues and, in spite of using material carefully prepared from the stainless steel, occasional tissue reaction of a moderate or severe degree has been found. In the absence of sepsis any such tissue reaction is taken as evidence of toxicity of the metal due to its dissolution in the body fluids.

It is clear that any method of increasing corrosion resistance and so reducing toxicity is of importance; this is especially true when the metal is used for the manufacture of hip cups and similar appliances, as well as for the more usual plates, nails and screws. The physical experiments have shown that contact between metal surfaces usually results in the transference of small fragments of metal from one surface to the other. This indicates that there may be a variable factor present in screwing, bolting and drilling which might act as a source of electromotive force and a cause of metallic corrosion. Observations have been made of the tissues surrounding buried orthopaedic appliances. Specimens were examined in order to compare the tissue reaction to metal handled with considerable force during its insertion (for example, screw heads and bolts) with that to metal not treated so severely (for example, the flat surfaces of plates and nails).

PATHOLOGICAL OBSERVATIONS

The tissues surrounding buried metal were examined during operations for the removal of instruments of internal fixation which had fulfilled their purpose. It was found that the tissue reaction varied from case to case. Sometimes marked "rusting" appeared to have occurred, the tissues adjacent to the metal being stained deep brown. Occasionally granulation

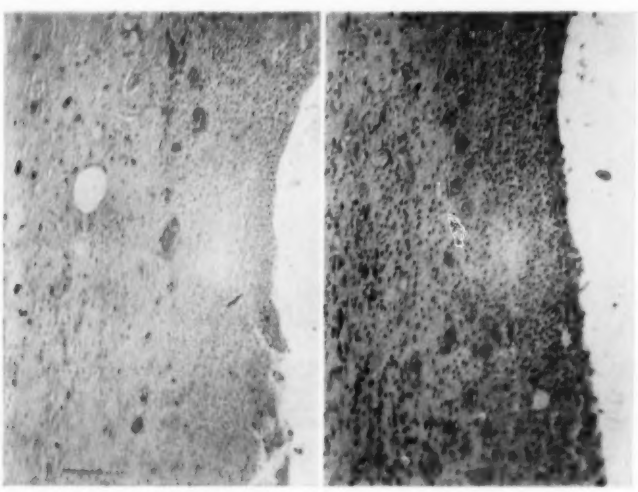


FIG. 11 FIG. 12

Case 7—Section of tissue that had been in contact with the flat surface of a plate for six months. There was no evidence of corrosion of the plate and the surrounding tissue was not discoloured. The sections show a thin scar with little cellularity. Treatment with Prussian blue showed no infiltration of iron (Fig. 12). (×240.)

tissue and dense fibrous tissue covered a bolt or screw head. In all cases the metal was surrounded by a shiny membrane. There was no evidence of infection in any of these cases, nor was any macroscopic corrosion of the metal observed.

Pieces of the tissues that had been in contact with F.M.B. steel were removed from a succession of fracture cases. Some were prepared with haematoxylin and eosin to show the cellular detail, and others were treated to demonstrate iron infiltration by the Prussian blue reaction. No cases with complications such as sepsis or Paget's disease, or in which more than one metal had been inserted, were included in our survey.

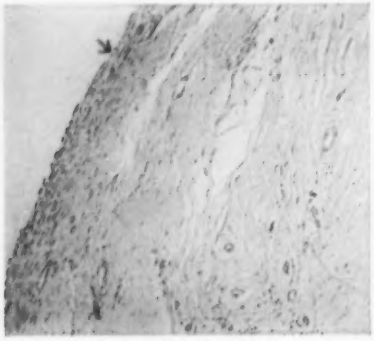


FIG. 13

FIG. 14

Case 12—Tissue in contact with screw head for three months. There was no macroscopic evidence of corrosion. The sections show a moderate cellular reaction in the scar, and the Prussian blue reaction showed fairly marked infiltration with iron. (Fig. 13—×80; Fig. 14—×330.)

FIG. 15

Case 15—Tissue in contact with a bolt head for three months. Macroscopically the tissue was heavily stained with brown pigment. The metal showed no corrosion. The sections show a marked vascular and cellular reaction, consistent with the presence of severe electrolytic inflammation. (×240.)

The pathological observations are summarised in Table V, and illustrative photomicrographs are shown in Figures 11 to 15.

The severe cellular reaction and the heavy iron infiltration seen in cases 2 and 15 (where the tissue had been in contact with a heavily handled bolt head (Fig. 15) was in marked contrast with the minimal reaction and almost complete absence of iron infiltration seen in Cases 1, 3, 5, 7, 9 and 10, in which the tissue had been in contact with those parts of plates and trifin nails which had not been handled with tools (Figs. 11 and 12). In an intermediate position between these two groups are Cases 4, 6, 8, 11, 12 and 13, which refer to tissues

adjacent to screw heads, and Case 14, in which the tissue had been in contact with the upper end of an intramedullary nail (Figs. 13 and 14). In these there is an abnormally great though varying amount of iron in the tissues and a marked though again varying cellular and vascular reaction (electrolytic inflammation). The presence of iron staining and electrolytic inflammation in the tissues adjacent to the metal is evidence of local corrosion of the steel,

TABLE V

CORRELATION BETWEEN SEVERITY OF HANDLING METALS AND DEGREE OF TISSUE REACTION AND OF IRON INFILTRATION AROUND THEM

Case number	Period buried (*months*)	Site of tissue	Degree of severity of handling during insertion	Degree of tissue reaction	Degree of iron infiltration	Relevant illustrative figures
1	3	Nail fin	None	None	None	—
2	3	Bolt head	Very severe	Severe	Very severe	—
3	3	Plate	None	None	None	—
4	3	Screw head	Mild	Some	Some	—
5	4	Plate	None	None	None	—
6	4	Screw head	Mild	Severe	Very severe	—
7	6	Plate	None	None	None	11–12
8	6	Screw head	Mild	Some	Some	—
9	4	Nail fin	None	None	None	—
10	3	Nail fin	None	None	None	—
11	4	Screw head	Mild	Some	Some	—
12	3	Screw head	Mild	Some	Severe	13–14
13	3	Screw head	Mild	Some	Some	—
14	12	Nail head	Severe	Severe	Severe	—
15	3	Bolt head	Very severe	Severe	Very severe	15

The bracketed pairs are from the same patient

even though macroscopic indication of this may not be visible on the metal. So far as can be judged these findings agree with those of Collins (1953) who found no undue reaction to F.M.B. stainless steel in tissues adjacent to Watson-Jones nails and to the shanks of screws.

The F.M.B. stainless steel material used in all these cases had been passivated in nitric acid and also by reverse plating or electrolytic polishing by the manufacturers. In spite of these efforts to render it as non-toxic as possible it has been shown that it can on occasion behave as a toxic metal, exciting a mild or severe reaction typical of electrolytic inflammation. This change of character appears to correlate with and be roughly proportional to the force used and the damage done to the metal during insertion.

ANALYTICAL EVIDENCE

Biopsy samples of tissues surrounding metal plates buried in man have been analysed spectroscopically for us by Dr A. H. Gillieson of the Atomic Energy Research Establishment, Harwell. The quantities found were compared with the normal concentration of the metals

in tissue (Tipton *et al.* 1953). All the samples were taken from regions adjacent to plates and nails which had been buried in the body for three to four months.

Two pieces of tissue were taken from the same patient: one from near a screw head and the other from the surface of an unhandled part of the plate. Both screw and plate were of stainless steel. These tissues correspond to biopsies 6 and 5 respectively. The concentrations of chromium, nickel, and manganese were very much greater near the metal which had been handled with tools, being six times the normal for manganese, twelve times for nickel, and over thirty times the normal for chromium. No other element was observed to be present in abnormal concentrations. The corresponding concentrations near the unhandled part of the plate, although high, were not significantly outside the normal range. A sample of tissue adjacent to a Vitallium screw head was analysed and found to have over twenty times the normal concentration of both cobalt and chromium; these are the two main constituents of Vitallium (Table I).

In none of these cases was any corrosion or tarnishing of the metal visible, yet the photomicrographs show and the analysis confirms quantitatively that the main constituent elements of the buried stainless metal are present in abnormally high concentrations in the tissues surrounding those parts of its surface which had been in contact with non-stainless tools, and that there is little, if any, excess concentration near the unhandled parts.

Although these results indicate a close correlation between the region of greatest concentration of metal in the tissues and the site of transferred particles, there is also clear evidence that the corrosion is not restricted to the foreign fragments alone but that the bulk of the buried metal is attacked. The quantitative results enable the amount of each element present in the tissues to be calculated, while the physical experiments indicate the mass of metal likely to be transferred from the tools during a given procedure. It is found that the amount of nickel and chromium present in the small sample of tissue analysed (0·1 gramme) was, itself, as great as the total mass of these elements usually transferred to the screw head from the driver. It is clear that the amount of metal passing into all the surrounding tissues is much greater than the total amount of tool metal adherent to the buried appliance (Bowden, Williamson and Laing 1954*b*). The high concentration of cobalt observed round the heads of Vitallium screws could not be caused by the corrosion of transferred steel particles but must be due to the dissolution of the Vitallium itself.

These measurements confirm quantitatively the picture suggested by examination of the stained microtome sections of tissue. They show clearly that considerable quantities of the so-called inert metals pass into the surrounding tissues; that this dissolution is greatest from regions which have been handled heavily with tools during the insertion of the appliance; and that the amount of metal passing into solution exceeds the total weight of the foreign fragments. The analysis of the tissue surrounding the Vitallium plate gives direct evidence that corrosion of the bulk metal occurs. These results provide strong support for the suggestion that the transferred fragments may act as corrosion centres and initiate a continued passage of the bulk metal into the surrounding tissues.

DISCUSSION

Because of the physiological dangers of electro-chemical dissolution great care is taken to ensure that only metals having a very high corrosion resistance are buried in the body and, also, that different metals are never inserted in the same patient. All orthopaedic nails, screws, bolts and plates are made from stainless materials, but the metal used for surgical tools does not have a high corrosion resistance. Tiny particles of the tool steel adhering to the buried metal may thus be very important. Much more serious than the dissolution of the transferred fragments themselves, however, is the fact that they can act as centres of corrosion and may initiate a continuing dissolution of the metal to which they are attached. Measurements

show that an appreciable difference in electrode potential can be set up between the steel of the tools and the metal used for the internal appliance, so that an electrolytic couple will be formed. The passivity of corrosion-resistant metals is due to the protection of their superficial oxide layer. There is evidence that with most metals transferred particles penetrate the oxide layer and adhere to the underlying metal. The dissolution of these embedded foreign fragments, if they are anodic, may lead to a serious breakdown of the oxide layer and to the onset of continuing corrosion; if the adherent particles are cathodic (the relative electrode potential is very dependent on the extent to which the oxide layer is distributed and also on the surrounding medium; see, for example, Evans 1946) a continuing reaction may again result. Thus the presence of apparently trivial fragments of non-passive metal may cause a continuous flow of metal ions into the surrounding tissues. Metal transfer may be expected to have an important influence on corrosion in the presence of a low extracellular pH as the phenomenon of anodic dissolution may then become important. A low pH (5·5 or less) follows all operations and trauma (Clay Ray Murray 1941) and is more marked and prolonged in the presence of sepsis (Fink and Smatko 1948).

These experiments have shown that many of the actions common in orthopaedic surgery are almost impossible to perform without leaving tiny fragments of the tool adherent to the buried metal. Metallic transfer has been demonstrated in all three procedures studied. The amount of transfer is very much dependent on the manner of handling the components and increases markedly if the tools slip at all. Autoradiographs show that the transfer is heaviest in those regions where great pressure is exerted and where much slip occurs.

In screwing and bolting the amounts observed varied between one and one hundred micrograms, depending on the metal used, the force applied, and the amount the tool was allowed to slip. The data relevant to an actual orthopaedic operation are probably those in which a large amount of slip occurred and very great force was applied. Electro-chemical considerations indicate, and the analytical results confirm, that these transferred particles may act as corrosion centres and lead to a continued dissolution of the bulk metal. They may be a cause of the variable tissue reaction observed around buried appliances, and in fact the pathological evidence has shown that the distribution of this reaction correlates closely with that of the transferred particles.

The amount of transfer occurring when a pilot hole is drilled into bone through a plate hole can be very much greater than that in either screwing or bolting and may well be a serious source of corrosion. If the twist-drill is allowed to rub against the shoulder of the plate hole while the pilot hole is being drilled the transfer can be of the order of milligrams. The surfaces of the shoulder of the plate hole and of the screw neck are particularly vulnerable to the onset of corrosion, for when the screw head is seated in a countersunk hole any corrosion pores which may develop in the oxide layer will not readily be closed by dissolved oxygen carried to the site, as the close fitting of the screw head and the countersink does not permit easy access to these surfaces. These large amounts of transfer, together with the subsequent differential aeration of the region, may be a cause of the gross corrosion which is occasionally observed at such points (Bowden, Williamson and Laing 1954*b*). The danger of this would be diminished if all pilot holes could be marked and drilled before the plate was attached. The metallic transfer direct to the bone itself from a twist-drill is not likely to be of great importance as the amount is small and decreases rapidly with the age of the drill.

CONCLUSIONS

The amount of metallic transfer depends on the specific nature of the contracting surfaces. There are several ways in which its control might be attempted. One simple way is to consider the relative hardnesses of the contracting metals. The effect of the hardness of the tool has been investigated quantitatively. Samples of surgical drivers and screws have been treated to alter

their hardnesses and the amounts of transfer occurring with various combinations have been measured. Table VI shows the relative transfer observed. These figures should be regarded as approximate only, because the amount of transfer showed large variations. All the samples of orthopaedic drivers and screws examined had hardnesses of about 150 and 380 V.H.N. respectively and the transfer under these conditions has been taken as unity. Clearly, the transfer might be reduced appreciably by increasing the hardness of the drivers or softening the screws.

TABLE VI

DEPENDENCE OF THE RELATIVE AMOUNT OF TRANSFER ON THE HARDNESSES OF DRIVERS AND SCREWS

	Steel driver 150 V.H.N.	Steel driver 300 V.H.N.
Steel screw, 380 V.H.N.	1	1/10
Steel screw, 133 V.H.N.	$\frac{1}{4}$	1/10

It does not necessarily follow, however, that the smallest electrolytic action will occur when a hard tool is used. Although a hard tool will reduce the metallic transfer, it will cause increased mechanical deformation of the oxide layer on the screw and may thus affect the inertness of the metal. Further experiments are necessary to determine the relative importance of these two factors.

If identical metals are used for the tool and screw the effect of the differential electrode potential should be reduced, but, conversely, the amount of transfer would be increased; other experiments have shown that this is much greater between similar metals (Rabinowicz and Tabor 1951). The point is clearly illustrated by the results for the transfer from steel to steel, and from steel to Vitallium, shown in Tables II and III. In view of the results given in these Tables it seems probable that the two steels are already behaving as like metals, and no great increase in transfer would be expected if the tools also were made out of steel. It should be remembered, however, that even if an adherent fragment has a composition identical with that of the bulk metal it may nevertheless have a different electrode potential due to the deformation it has undergone. Again, further experiments are necessary to decide the relative importance of these factors.

Metallic transfer could be reduced appreciably if a suitable inert lubricant were used between the tools and the orthopaedic components. This might provide a simple solution to the problem and could be especially effective in reducing transfer if used in conjunction with non-slip screwdrivers. This approach is worthy of closer investigation.

This work emphasises the importance of avoiding whenever possible any metallic contact with the metal to be buried, particularly when sliding or large forces could occur, or when the contaminating metal is not stainless. This, of course, applies to the storage of components as well as to their insertion. The metal screw set holders in common use ought to be considered with this in mind. The sliding between screw and the edges of the slot of the holder which takes place when the selected screw is removed might easily cause particles of the holder to become attached under the neck of the screw. The protective oxide film on the screw could also be damaged by this friction.

The quantity of metal transferred from the tools used has been measured in several common orthopaedic procedures, and the level of tissue reaction and the concentration of metals normally found in the tissues surrounding buried appliances have been studied pathologically and analytically. It has been demonstrated that these quantities are closely correlated. The greatest pathological effect is found near regions of the buried metal which

received most metal from the orthopaedic tools, and there is little or no reaction near the parts of the metal surfaces which were not touched.

Adherent fragments of foreign metal may exert a profound influence on the surface properties of the metal to which they are attached. While they are not the only cause of the corrosion of orthopaedic components they may nevertheless be an important one and merit careful consideration. These observations suggest that in orthopaedic surgery attention should not be focused on the buried metal alone. The transfer to it from the tool may be important, and it is the particular combination of the tool and metal used in the operation which must be considered.

SUMMARY

1. Experiments using radio-isotope techniques show that whenever an orthopaedic appliance is inserted into the body tiny fragments of the instruments used to handle it become detached and welded on to the surface of the buried metal.

2. There is evidence that these particles of non-passive tool-metal may cause a small but continuing dissolution of the stainless metal to which they are attached, and that this dissolution, although too small to be detected as visible corrosion, may produce an adverse tissue reaction.

3. In samples of human tissue taken from sites adjacent to buried metal the greatest level of tissue reaction and the highest concentration of iron were found near those parts of the appliances that had been handled with the tools and received the greatest amount of transferred metal.

4. Spectroscopic analyses of similar samples confirmed that the concentration of metal in the tissues surrounding a buried appliance correlates closely with the distribution on its surface of transferred tool metal. Particularly high concentrations of chromium, nickel, and cobalt were found near regions where the transfer had been heavy. The dissolution was not limited to the non-passive foreign metal; the stainless appliance was also dissolved in those regions.

5. The influence of the manner of handling the tools on the amount and distribution of transfer has been studied, and suggestions are made whereby the transfer might be reduced.

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THE HISTOCHEMISTRY OF THE PREOSSEOUS STAGE OF BONE REPAIR STUDIED BY AUTORADIOGRAPHY

The Effect of Cortisone

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Studies on the histochemistry of bone repair have been limited in the past to the stages of calcification and ossification. In previous work the pH in the fracture haematoma, the accurate localisation of the enzyme alkaline phosphatase and the deposition of calcium phosphate complexes have been examined, mainly by the use of special stains and techniques. A historical survey of these investigations has been given in a previous paper (Duthie and Barker 1955).

To obtain further biochemical information, various pathological conditions have been induced in experimental animals. For example, in scorbutic guinea-pigs, Bourne (1948) studied the alteration in the alkaline phosphatase content of regenerating skull bone after fracture, and in animals deficient in vitamins A and D and in others given excess of vitamin D, Copp and Greenberg (1945) related the degree of calcification to the breaking strain of the healing fracture.

The effects of hormonal imbalance occurring in rickets or after the injection of insulin or oestrogen have been studied (Stuck 1932, Pollock and Ghormley 1941). The healing of fractures was affected by these hormonal factors, although it proceeded independently of such abnormal nutritional conditions as starvation and protein deficiency (Armstrong 1944).

Although the histological and clinical descriptions of the stages of healing—that is, procallus, fibrocartilaginous callus and bony callus—are well known (Enneking 1948, Urist and McLean 1941), from the histochemical point of view it is of more value to consider these stages morphogenetically (Needham 1952) as wound closure, demolition and removal of damaged cells, and dedifferentiation of cells to provide new tissues by formation and growth of blastemata. Under the stimulus of trauma, in bone repair the blastemata arise both from periosteal and medullary cells to form the fibrocartilage or callus. This stage, during which the mucopolysaccharide complex—chondroitin sulphuric acid—is metabolised and used by the developing cartilaginous masses, is of particular interest and importance. This mucopolysaccharide with its ester sulphate grouping can be studied by "labelling" it with radioactive sulphur and by staining it metachromatically with certain dyes. Moreover, this form of histochemical investigation gives information about the possible origin and function of the cells during the process of differentiation. The secretion of chondroitin sulphuric acid forming their most intimate environment enables these cells to be identified as well as reflecting their internal economy.

In a previous investigation (Duthie and Barker 1955) we were unable to demonstrate the localisation of ^{35}S in the fibrocartilaginous callus stage of a healing fracture site, although it was shown that cartilage cells elsewhere used this isotope and stained metachromatically. This was attributed to the inadequate circulating blood level of ^{35}S at the time when the chondrocytes in the callus were assimilating sulphur. To attain a sufficiently high concentration of ^{35}S in the circulation at each stage of the healing process, it was found necessary to inject the radioactive solution twenty-four hours before killing the animal.

Many reports of the effects of cortisone and ACTH on various diseases and biochemical processes have been published (Hensch *et al.* 1949, Layton 1951). Since the description

by Ragan *et al.* (1949) of the delay in the healing of decubitus ulcers and operation wounds in patients with rheumatoid arthritis and erythema multiforme receiving ACTH, much experimental work has been undertaken to determine the inhibitory effect of these hormones, especially in diseases of connective tissue and in the repair of wounds and fractures. However, there is little information about the effect of cortisone on the histochemistry of these tissues.

In rabbits receiving cortisone, Blunt *et al.* (1950) described the histological features of the delayed healing of experimental fractures. Sissons and Hadfield (1951) demonstrated radiographically and with more detailed histological examination the inhibition by cortisone of the repair processes—particularly the development of the vascular granulation tissue—and the resultant effect on the fracture callus, with emphasis on the different reactions in differing species of experimental animals.

To add to the general uncertainty about the effect of cortisone on the healing of fractures, in rats receiving this hormone Key *et al.* (1952) were unable to demonstrate any delay.

In this study the effect of the administration of cortisone on the histochemistry of a healing fracture in the rat will be described, and additional information will be presented on the histochemistry of the normal repair of bone.

METHODS AND MATERIALS

Two separate series of animals were examined. In the first, sixteen five-week-old albino rats, of both sexes, weighing approximately 70 grammes each, were used. Under ether anaesthesia the left tibia of each animal was fractured with bone-cutting forceps at the junction of the uppermost third and middle third. Eight of these rats were kept as controls. One of the remaining eight, weighing 70 grammes, received an intraperitoneal injection of 0·7 millilitres of sterile normal saline containing 2,100 μc of ^{35}S (30 μc/gramme body weight) supplied by the Radiochemical Centre, Amersham, as carrier-free sulphate in aqueous solution. Twenty-four hours later this radioactive rat and a control rat were killed with chloroform and the fractured tibia of each was immediately dissected out of the soft tissues and placed in 10 per cent neutralised formalin.

The remaining fourteen rats were kept and killed at various intervals after operation; each animal in the radioactive group was weighed and given an intraperitoneal injection of 30 μc/gramme body weight of ^{35}S, twenty-four hours before death. The subsequent processing of the bones was carried out as already described by one of us (Duthie 1954).

Microautoradiographs, macroautoradiographs and radiographs were prepared from all the tibiae. The techniques used have been fully described (Duthie and Barker 1955).

In the second series twenty similar rats were used. They were subjected to the same operative treatment as the first group, but in addition each rat received, until the day of death, a daily intramuscular injection of 1 milligram/50 grammes body weight of a sterile saline suspension of cortisone acetate. On this dosage all the rats failed to gain normally in weight and size; this effect was also observed by Sissons and Hadfield (1941).

One rat died after nine days, another after sixteen days and two after twenty-one days, with marked cachexia, nasal discharge and diarrhoea; histologically there were changes in the suprarenal glands of these animals.

In the second series the injected dose of ^{35}S was reduced to 20 μc/gramme body weight because the autoradiographic images obtained in the first series were sufficiently dense after only two weeks' exposure.

RESULTS

First Series (without cortisone)

At twenty-four hours—The cortical fragments of mature bone with adult bone cells in the lacunae, the marrow cells and haematoma formation were the main components of the fracture with the ruptured layer of the periosteum falling into the defect accompanied by some fibrous tissue (Fig. 1). These periosteal cells were of two main types: the outer layer cells were

typical fibroblasts, whereas those of the inner layer had elongated, pale cytoplasm and oval basophil nuclei—osteogenic cells. A normal distribution of mast cells was seen in the outer

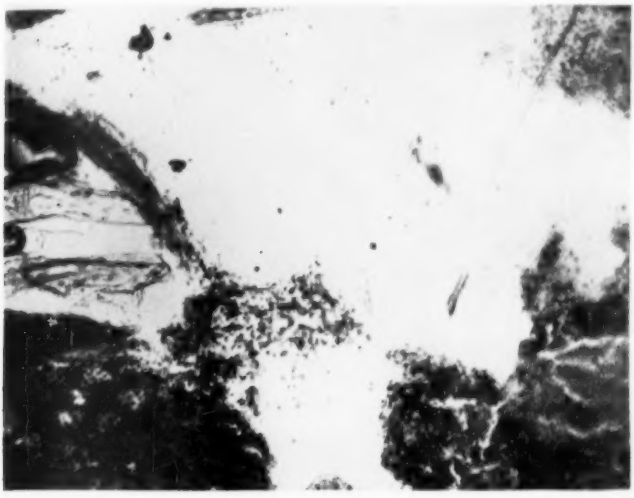

FIG. 1

Section through fracture defect of twenty-four hours' duration showing the periosteum falling in from the left, across fragmented cortex; below this there are bone marrow cells with red blood cells forming a haematoma on the right. (×100.)

FIG. 2

Section twenty-four hours later, which shows the fracture and the medulla containing numerous mast cells on the left. Between these two areas there is a fan-shaped area consisting of differentiating osteogenic cells. (×100.)

layer. No definite metachromasia was seen and there was no evidence of the presence of ^{35}S.

At forty-eight hours—There was marked hyperplasia and dedifferentiation of the inner or cambial layer of the periosteum. As well as marked local proliferation, the elongated cells

had changed to a globular shape, especially in the vicinity of the fracture site, as they now exhibited metachromasia. In both intercellular and intracellular situations ^{35}S was present.

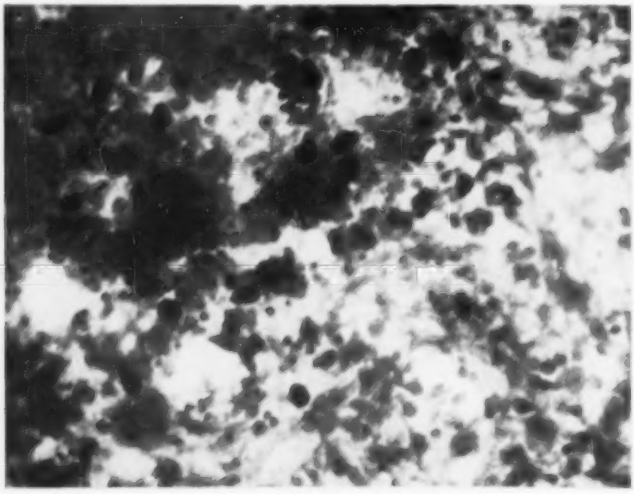

FIG. 3

Section similar to that shown in Figure 2, with numerous mast cells lying in the bone marrow and the differentiating osteogenic cells on the right. (×400.)

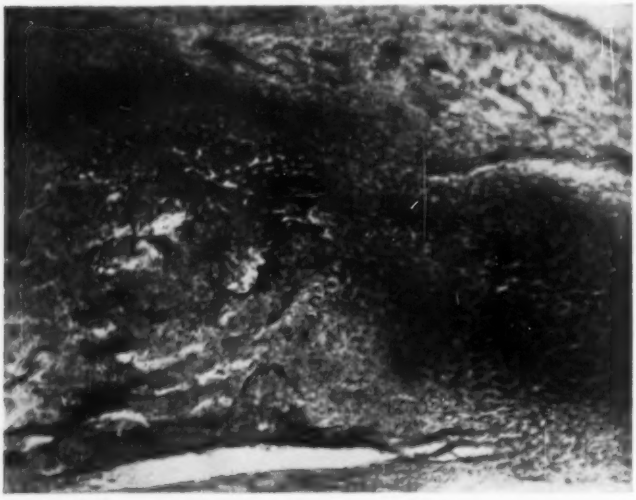

FIG. 4

Section through the subperiosteal area showing marked hyperplasia and differentiation of cells of the cambial layer with secretion of darkly staining metachromatic substance around them after ninety-six hours. (×100.)

These features did not cease abruptly but penetrated some little distance along the periosteum on each side of the fracture line.

Within the medullary cavity there was dedifferentiation of cells to form cells possessing

an elongated, pale cytoplasm with large round basophilic nuclei, similar to osteogenic cells and forming the medullary blastema. Mast cells were numerous among the bone marrow cells adjacent to this area (Figs. 2 and 3).

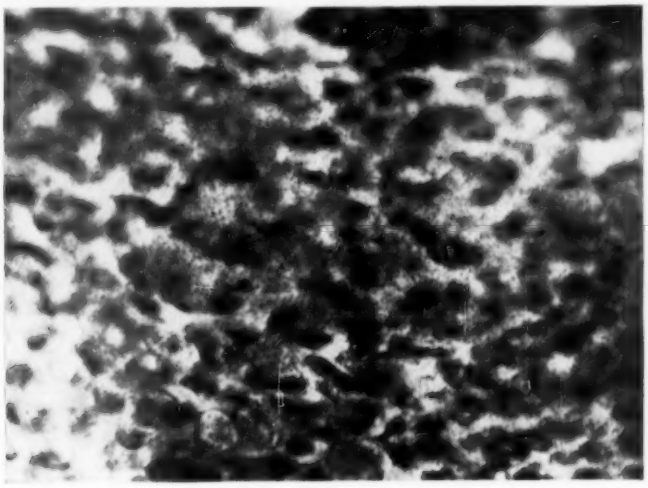

FIG. 5

Section of same area to show the cells becoming globular in shape and exhibiting a diffuse and high grain density from ^{35}S. (×400.)

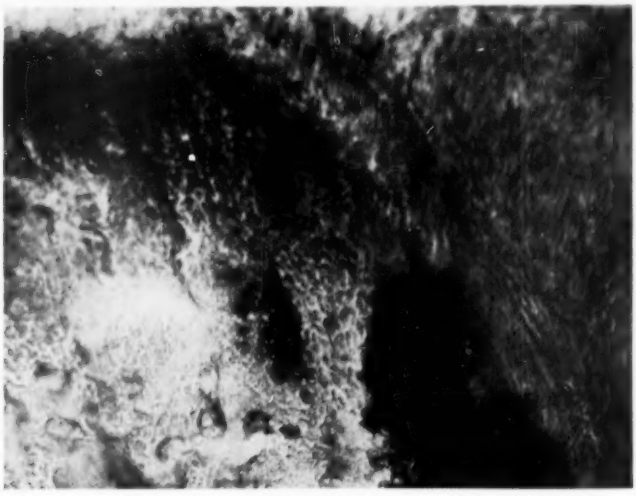

FIG. 6

Section, at seven days, to show sweeping in of the differentiating and thickened periosteum from the top left corner over the cortical bone. There is also formation of a cartilage blastema which appears dark because of the metachromatic stain and grains resulting from its ^{35}S content. (×100.)

At ninety-six hours—All these features were more marked (Figs. 4 and 5).

At seven days—The cellular dedifferentiation had progressed to form the typical periosteal blastema with aggregation and orientation of mature chondrocytes exhibiting obvious

metachromasia and having a high content of ^{35}S (Figs. 6 and 7). This was most marked adjacent to the fracture line. Mast cells were still evident, and in one particular area in this

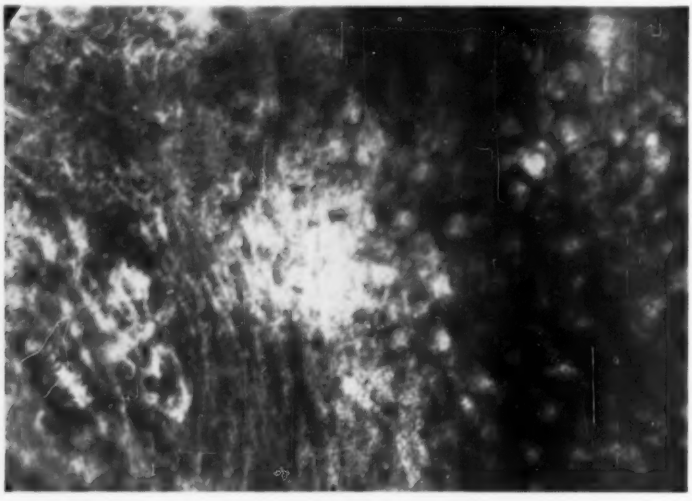

FIG. 7

Section through same area showing the formation of mature chondrocytes which are exhibiting metachromasia and a high content of ^{35}S. (×400.)

FIG. 8

Section of fracture line which shows a nest of mature chondrocytes, exhibiting metachromasia and around which there are areas of amorphous, metachromatic substance, at ten days. (×100.)

rat there was an abnormal number around a cartilaginous mass lying in the differentiating mesenchymal cells.

At ten days—The periosteal blastema consisted of several nests of adult chondrocytes (Fig. 8) and between these there were small collections of a metachromatic, amorphous, non-cellular substance. In one area there was occurring typical endochondral ossification of cartilaginous

blastema with the phases of chondrocyte development and degeneration, and formation of trabeculae of cartilaginous remnants which were surrounded by osteoblasts and vascular

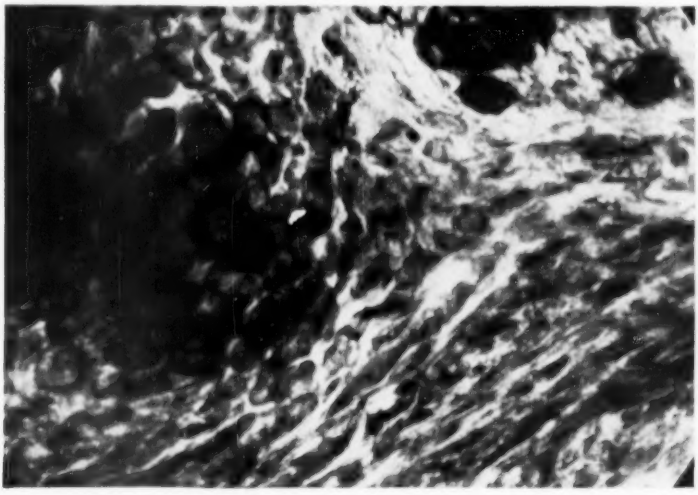

FIG. 9

Section in same area showing differentiation of the elongated osteogenic cells into adult chondrocytes surrounded by rings of dark metachromatic stain. (× 400.)

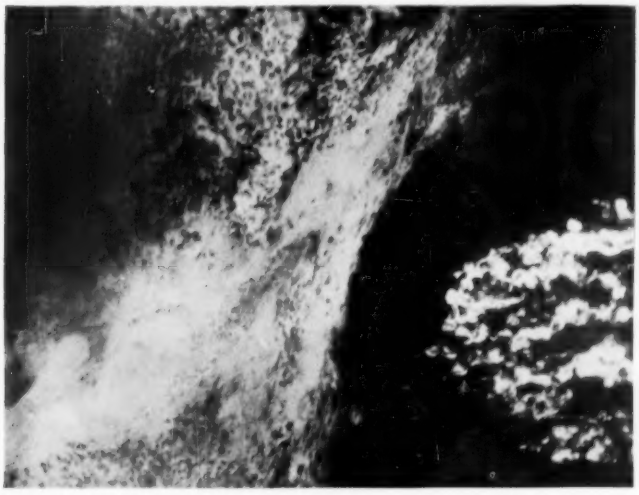

FIG. 10

Section through an area of cartilage blastema which is undergoing endochondral ossification with trabeculae formation and in between which there are osteoblasts. To the immediate left of this, there is an area of amorphous metachromatic intercellular substance. (× 100.)

tissue. Bone marrow cells had given rise to calcified trabeculae, and mast cells were still numerous among the cells of the marrow cavity (Fig. 10). In another area of this section there was continuing dedifferentiation of the mesenchymal cells into cartilage.

At fourteen days—This picture was more advanced and the amorphous metachromatic material

was more abundant and contained radioactive sulphur (Fig. 11). Numerous mast cells containing ^{35}S were present, particularly in the periosteal layer.

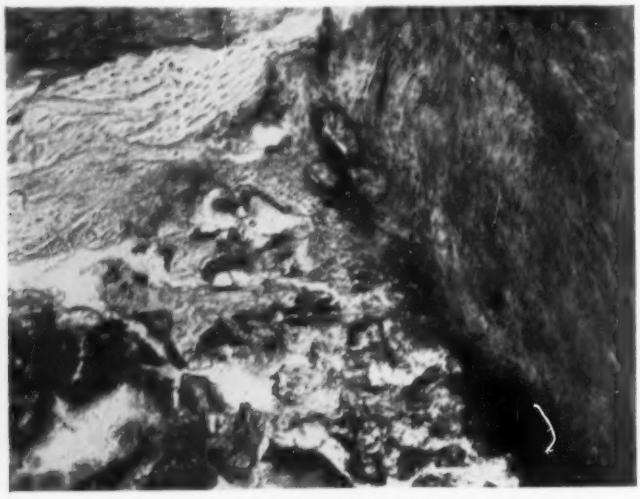

FIG. 11

Section after fourteen days showing calcified trabeculae below cortical bone on left of picture. Differentiating cells are to be seen sweeping downwards over this area and staining metachromatically. (×100.)

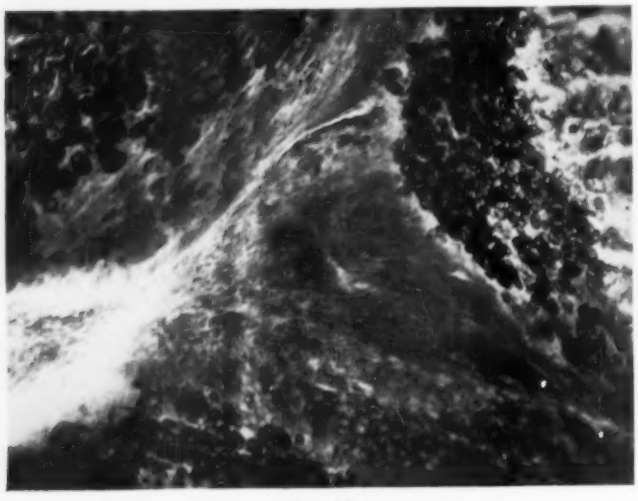

FIG. 12

Section, twenty-one days after fracture, through three areas of endochondral ossification between which there is amorphous, intercellular material. Mesenchymal cells are sweeping into this space. (×100.)

At twenty-one days—There were three areas of endochondral ossification (Fig. 12) which were metachromatic, contained radioactive sulphur (Fig. 13) and led to new bone formation. Mesenchymal cells from the periosteum were still seen sweeping into the fracture area and differentiating. Mast cells were no longer present.

At twenty-eight days—New bone formation was marked and only a thin zone of fibrous tissue and cartilage remained in the fracture area.

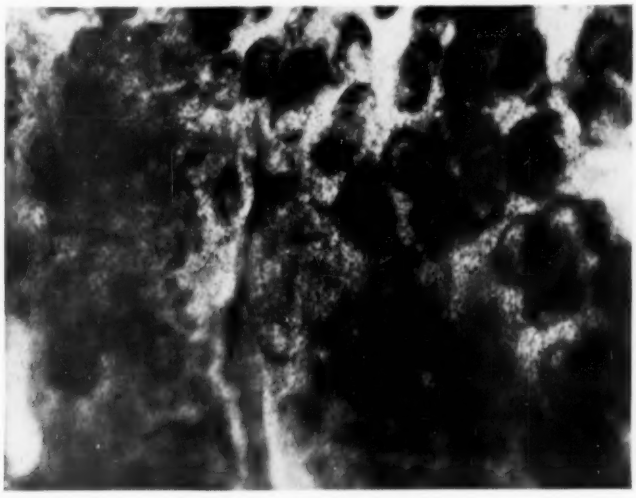

FIG. 13

Section through one of the above areas to show mature chondrocytes surrounded by rings of metachromatic intercellular substance and containing radioactive sulphur. (× 400.)

FIG. 14

Section, at ninety-six hours, of the periosteal layer with mature cortex below, to show proliferation and differentiation of cells of the cambial layer to form a cartilage blastema exhibiting metachromasia. (× 100.)

Second (cortisone-treated) Series

At twenty-four and forty-eight hours—The cellular components in the fracture were similar to those in the first series. The epiphysial plate appeared normal, and metabolised sulphur as in our previous investigation (Duthie and Barker 1955).

At ninety-six hours—Proliferation and differentiation of the inner layer of the periosteum had taken place to form the cartilage blastema of the periosteum (Fig. 14), and there was evident metachromasia and radioactivity over large areas of amorphous material. In the

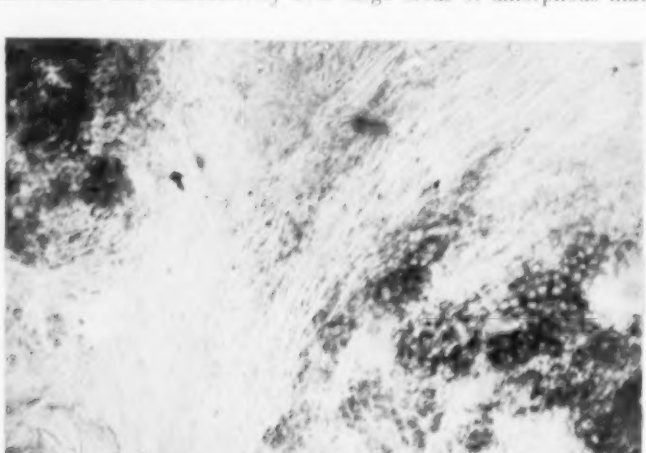

FIG. 15

Section of similar area, three days later, which shows formation of cartilage nests from differentiating cells sweeping into the centre. The cartilage cells, although exhibiting metachromasia and radioactivity, lack cellular detail. (×100.)

FIG. 16

Section from the same animal of the epiphysial region to show narrowing of the cartilage plate with increased fragmentation and irregular formation of the trabeculae. (×100.)

epiphysis the cartilage plate was reduced in depth by the disappearance of the phase of hypertrophying chondroblasts into chondrocytes, and this was accompanied by an increase in the intercellular matrix. The content of ^{35}S was high. The trabeculae appeared more fragmented and irregular and the bone marrow cells and osteoblasts extended higher into

the degenerating mass. The osteoblasts were reduced in number. At the fracture site there was differentiation within the bone marrow cells to form cells with a pale blue cytoplasm and large dark blue nuclei lying within a framework of irregular columns containing small cyst-like

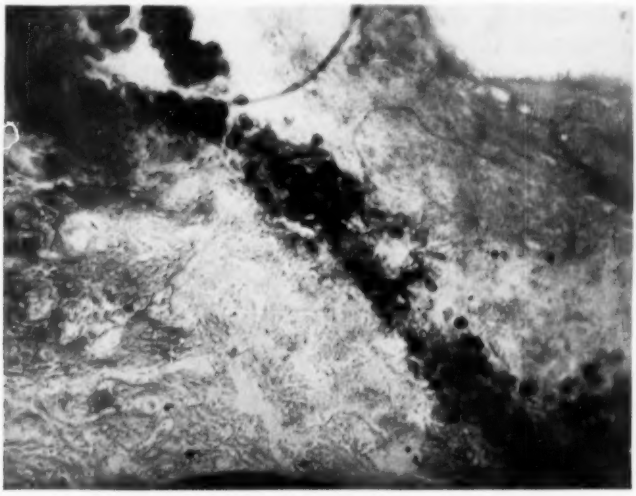

FIG. 17

Section of fractured end of bone on left with cartilage cells lying across fragment. Outside this and separating it from a cyst, there is abundant amorphous material, staining metachromatically after ten days. (×100.)

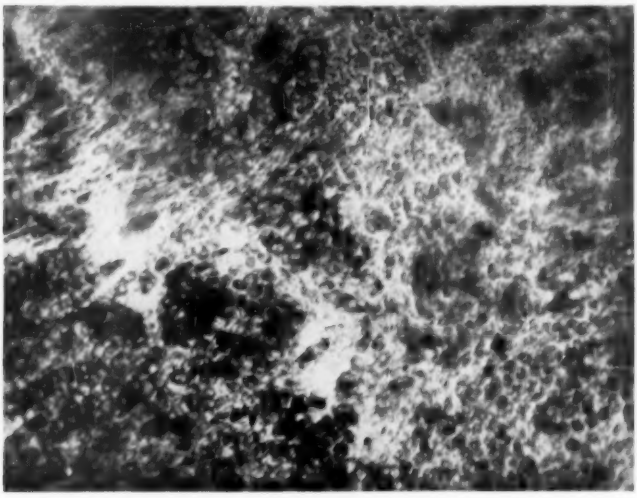

FIG. 18

Section through the cartilage blastema to show the loss of structure and emptying of cellular contents to become vesicular in appearance. (×100.)

spaces. This area was neither metachromatic nor radioactive and lay between the cells of the bone marrow and the fracture site, whose wall possessed some differentiating metachromatic cells. There were mast cells in the marrow.

At seven days—There was typical differentiation to form nests of cartilage cells which were

both metachromatic and radioactive (Fig. 15), but in one area the cartilage cells were without nuclei and amorphous material was abundant. Mast cells were increased in number in the bone marrow and the area between the marrow and the fracture defect was more evident. The epiphysial plate was similar to that seen at ninety-six hours (Fig. 16).

At ten days—There were large areas of cartilage nests without any evidence of endochondral ossification. In these nests the cartilage cells lost detail and appeared as small empty vesicles among abundant amorphous metachromatic material (Figs. 17 and 18). In the region of differentiating bone marrow cells there was an area of amorphous material but the remainder was osteoid tissue.

At fourteen days—This picture persisted and across the fracture line there was now a zone of fibrous tissue composed of mature fibroblasts which showed no evidence of differentiation.

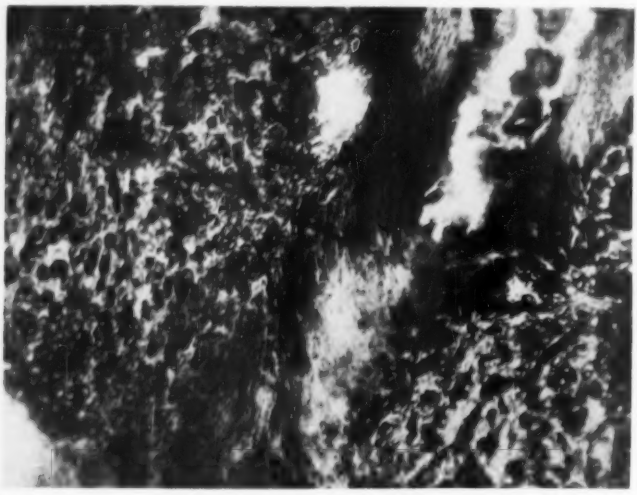

FIG. 19

Section at twenty-one days through fracture line which now consists of mature fibroblasts and free amorphous material, and separates two areas of cartilage blastemata. There is no evidence of the process of endochondral ossification. (× 100.)

At twenty-one days—The appearance was similar except that new bone had been laid down on each side of the fibrous tissue zone and small islets of cartilage were seen (Fig. 19). No endochondral ossification was observed.

MACROAUTORADIOGRAPHY AND RADIOGRAPHY

First Series (without cortisone)

At twenty-four hours (Fig. 20)—The distribution of ^{35}S throughout the various components of the tibia was similar to that described in our previous paper (Duthie and Barker 1955), with no accumulation in the fracture defect and less in the distal than in the proximal fragment.

At forty-eight and ninety-six hours (Fig. 21)—The pattern was similar to that at twenty-four hours.

At seven days (Fig. 22)—A large amount of radioactive sulphur was deposited in and around the fracture. The radioactive area was globular in shape and extended in both directions along the periosteal and medullary regions of the shaft. In the radiograph there was evidence of early deposition of opaque calcium salts around the shaft above and below the fracture

corresponding closely to the shape of the radioactive area but much less in extent. This shape is related to the lifting of the periosteum during the process of ossification.

At ten days—No further change had taken place.

At fourteen days—There was still a considerable deposition of ^{35}S around and distal to the fracture, but between the fractured ends of the shaft the amount was reduced. The characteristic shape was maintained and within the area further deposition of calcium salts had taken place from the bony callus.

At twenty-one days—The deposition of ^{35}S was less extensive and the globular shape of radioactivity was lost. This may have resulted from the lack of alignment of the fracture fragments since the cortex of the distal shaft was opposed to the fractured end of the proximal fragment. In the radiograph the calcium phosphate complexes appeared to be laid down in the areas already containing ^{35}S and therefore, presumably, chondroitin sulphuric acid.

At twenty-eight days (Fig. 23)—Union was nearly completed with marked bony callus formation, especially on the convex side and around the fibular defect. Very little ^{35}S was present in the fracture, but a radioactive zone surrounded a cyst-like area adjacent to the fracture line.

Second (cortisone-treated) Series

During the first forty-eight hours (Fig. 24)—The distribution of ^{35}S was similar to that of the first series but the epiphysial plate was much narrower and the trabeculae were less evident. The articular cartilage was also reduced in depth.

At ninety-six hours (Fig. 25)—The deposition of ^{35}S was marked in and around the fracture site, in the periosteum and medulla and also around the fibular defect. The other features were similar to those seen at twenty-four and forty-eight hours.

At seven days (Fig. 26)—The fracture defect was more pronounced and the radioactive sulphur was concentrated in the margins of the fracture area, between which there was an obvious space.

At ten days—The fracture site was sealed off by the typical globular mass of radioactive tissue. The thinness of the articular cartilage and epiphysial plate was most marked. From the radiograph evidence of the early deposition of small amounts of calcium salts was obtained.

At fourteen days—Only narrow radioactive zones remained along each margin of the fracture line, which was being gradually closed by further deposition of calcium salts.

At twenty-one days (Fig. 27)—Some ^{35}S was present in the vicinity of the fracture line, which was still evident on the radiograph. Calcium salts were laid down along the periphery of the globular mass but were less dense than was normal at this stage.

DISCUSSION

The histology of the early phases of repair—During the repair of these fractures, the process of wound closure was seen to consist of haematoma formation, with the periosteal layer and any adherent fibrous tissue or muscle falling into the defect. Organisation of the haematoma into granulation tissue was not evident and the arrest of bleeding probably occurred as a result of pressure on the ruptured vessels as well as by the active process of constriction of the vessel walls. In this series the interposition of muscle between the fragments was not usually seen because the fracture was produced after exposure of the shaft of the tibia by open operation.

" Demolition " or removal of damaged cells by any form of phagocytosis was not evident, and indeed there was no sign of actual damage to cells such as the osteocytes of the cortical lacunae. But some mechanism of stimulus must have been present, because dedifferentiation of the surrounding cells had occurred after the injury, and within forty-eight hours marked cellular change was taking place.

There were two main processes of repair originating in two different areas. Among the periosteal cells, both the undifferentiated mesenchymal cells and the differentiated osteogenic

FIG. 20

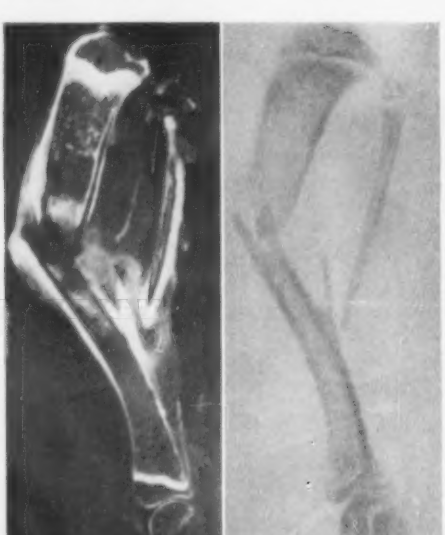

FIG. 21

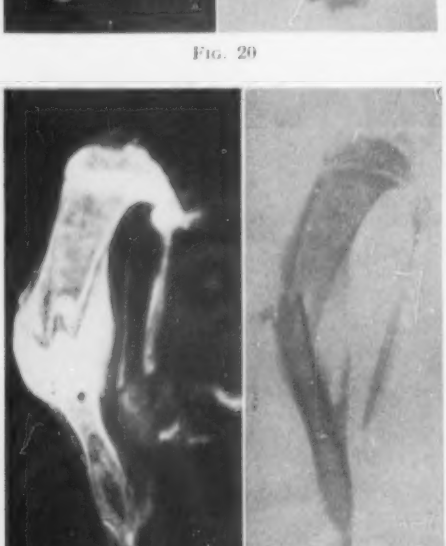

FIG. 22

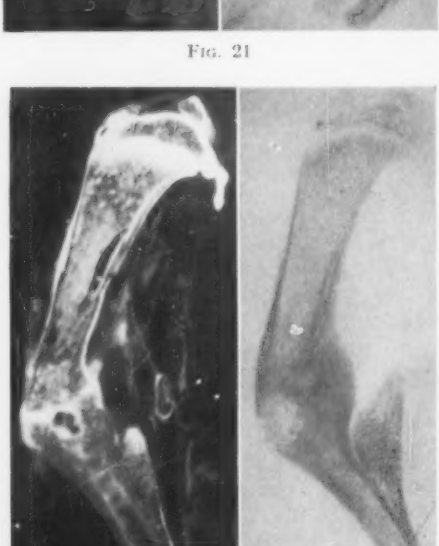

FIG. 23

Enlarged prints (× 3) of macroautoradiographs, with ^{35}S seen as white areas in fractured tibiae, compared with enlarged radiographs (× 3) of the same bones (right of each figure). Figure 20—twenty-four hours after fracture—The autoradiograph shows that although the various areas in bone, such as the epiphysial plate, articular cartilage, both layers of periosteum and shaft, have a high content of radioactive sulphur, there is no deposition around the fracture site. There is a higher density over the proximal fragment on comparison with the distal fragment. Figure 21—ninety-six hours after fracture—The autoradiograph is similar to Figure 20 except that there is increased deposition of ^{35}S in the soft tissues around the fracture site. Figure 22—seven days after fracture—There is a marked deposition of radioactive sulphur in and around the fracture defect, forming a globular mass. In the radiograph there is early deposition of calcium salts subperiosteally. Figure 23—twenty-eight days after fracture—There is the typical deposition of radioactive sulphur in all structures except for a thin line across the healing fracture site which has become broader and contains a cyst-like area. The radiograph shows formation of the bony callus.

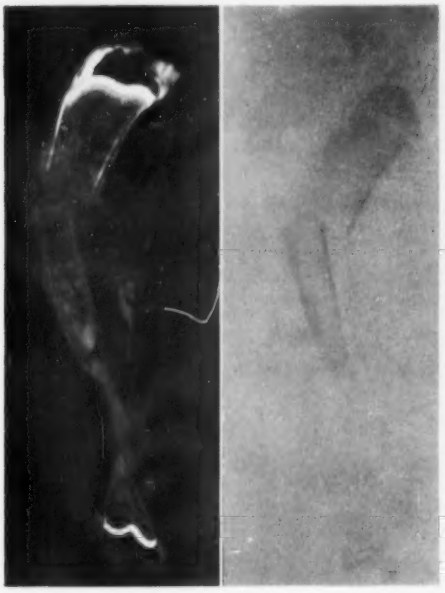

FIG. 24

FIG. 25

FIG. 26

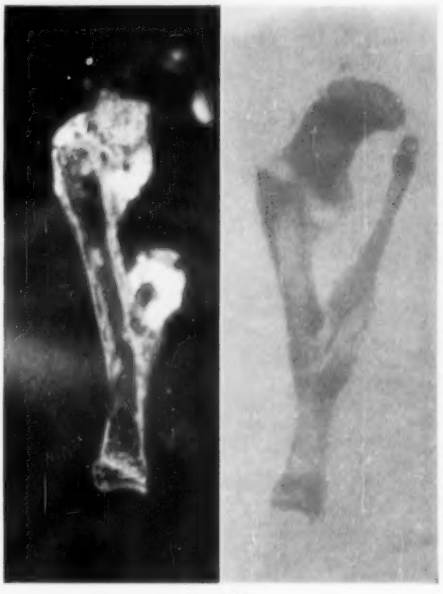

FIG. 27

Enlarged prints (×3) of macroautoradiographs with ^{35}S after cortisone, in fractured tibiae, compared with enlarged radiographs (×3) of the same bone (right of each figure). There is less deposition of radioactive sulphur in all areas with narrowing of the epiphysial plate. Figure 25—ninety-six hours after fracture—There is a marked deposition of ^{35}S in the fracture area and especially in the soft tissues, and this gradually reduces during the next three days (Fig. 26) leaving an obvious fracture defect. Figure 27—twenty-one days after fracture—The deposition of radioactive sulphur is obvious but irregular and patchy in appearance. In the radiograph the fracture line is still evident and the deposition of the calcium salt lacks density and appears to outline the callus.

cells of the cambial layer, there were changes in size, shape, number and orientation to form the cartilaginous blastema. Blood vessels were not seen until the familiar picture of endochondral ossification appeared after seven days. At this stage there were the usual five phases of chondrocyte development and final disintegration with trabecular formation, between which primitive blood vessels and osteoblasts could be distinguished. These osteoblasts did not appear to have formed within the blastema but rather had migrated inwards from surrounding structures, probably from the endosteal lining of Haversian canals in the cortex, from the inner layer of the periosteum and from the medulla. This process of differentiation with appearance of the osteoblast appears to be independent of the formation of the cartilage blastema.

From within the medulla there was differentiation of cells to form osteoblasts, later trabeculae of calcified tissue, and finally cancellous bone. The exact origin of these cells is uncertain because they could arise either from the undifferentiated mesenchymal or from the reticular cell. This process is a typical example of intramembranous ossification.

These two processes, although originating in different areas, did not remain separate to form the external (or periosteal) and internal (or endosteal) calluses, as has been described by Ham (1953). There was intermingling between the fracture fragments, and the cartilage blastema usually developed between the two areas of the medullary blastema.

The histochemistry of the early phases of repair—In the macroautoradiographs of the first three rats of the first series there was a greater amount of ^{35}S in the proximal fragment than in the distal. This may have been due to the interruption by the fracture of the blood supply from the periosteal and nutrient vessels. Within forty-eight hours the differentiating cells of the deeper layer of the periosteum adjacent to the fracture area had secreted around themselves a metachromatic substance containing radioactive sulphur. This reaction increased until the seventh day, when, accompanying the formation of the cartilaginous and medullary blastemata, the concentration of radioactive sulphur was most marked, particularly in the macroautoradiograph (Fig. 22). This was due mainly to the deposition and use of ^{35}S by the developing cartilage cells and also by all the differentiating osteogenic cells in this area. In the actual cartilaginous blastema radioactive sulphur was present in both an intracellular and intercellular situation, similar to that seen in the epiphysial region.

Because this secreted intercellular substance was labelled with ^{35}S and stained metachromatically, it is assumed to be the sulphated mucopolysaccharide, chondroitin sulphuric acid. In a recent biochemical review (Sarnat and Laskin 1954) the intercellular cartilage matrix was described as being composed of collagen, chondroalbumin and a glycoprotein, chondromucoid, which contains chondroitin sulphuric acid. It has been shown that the ester sulphate in this mucopolysaccharide can be labelled with radioactive sulphur (Dziewiatkowski 1952, Boström 1953) and although other substances are metachromatic, depending on their state of polymerisation, in cartilage metachromasia is regarded as specific for chondroitin sulphuric acid, because this is the only mucopolysaccharide which has been isolated from cartilage.

A similar intercellular substance is considered to be secreted by the osteogenic cells in both the periosteum and endosteum (Ham 1953). Under normal conditions we have demonstrated the use of ^{35}S in these areas and have shown that it is greatly increased in amount around a fracture, with obvious metachromasia.

This localisation of ^{35}S can be correlated to the distribution of the enzyme alkaline phosphatase in both chondrogenetic and osteogenic tissues and in their blastemata, which has been described by Pritchard and Ruzicka (1950). They observed that this occurs well in advance of calcification and therefore suggested that this enzyme had some other function as well as that concerned with the process of calcification. Siffert (1951) also noted that alkaline phosphatase was present among developing cartilage cells and therefore probably played some part in the growth of the cartilage matrix as well as in the process of calcification.

Layton (1950) showed by an *in vitro* tissue culture technique that regenerating muscle used more ^{35}S than normal muscle, and later Lacroix (1954) showed that the isotope was used *in vivo* during osteogenesis within the Haversian canals in the bone of dogs. In this study there is demonstrated for the first time the use *in vivo* of ^{35}S by differentiating cells of a fracture.

Factors initiating the repair process—In the vicinity of a fracture there is marked differentiation of cells with altered metabolism, and in the early preosseous stage of repair this reaction is avascular in nature. The factors which initiate this reaction are unknown. Much research, especially in Scandinavia, has been directed towards proving the existence of and identifying specific osteogenic substances which induce bone formation (Bertelsen 1944). This has been recently reviewed in detail (Lancet 1952).

In association with this local phase of repair we found an accumulation of mast cells both in the medulla and in the periosteum adjacent to where the blastemata were forming. We have described previously in the normal rat the presence of mast cells exhibiting metachromasia and containing ^{35}S in these sites (Duthie and Barker 1955). Their sudden appearance at forty-eight hours, their persistence for the next fourteen days and their accumulation near a fracture site suggest some possible function or reaction to the stimulus of trauma. Since their discovery by Ehrlich they have been observed to fluctuate in number and even in structure under various pathological conditions (Riley 1954). Their accumulation in connective tissue has been related to lymphatic blockage or to rupture of blood vessels, but this does not necessarily apply in our series, because the aggregation of cells was adjacent to rather than in the fracture defect. Indeed, in one particular area they were actually within an area of differentiating mesenchymal cells beside an early cartilage blastema.

The histochemistry of these cells has been recently reviewed (Asboe-Hansen 1954, McManus 1954) and it is considered that they possibly secrete either a heparin-like precursor substance, hyaluronic acid or histamine. The last substance would play an important role in the demolition phase of healing as well as in defence by altering the local vascularity and the migration of cells, and may be the reason for the accumulation of mast cells near a fracture. But Bensley (1950), from a histological study of changes in ground substances containing mast cells, has suggested that they may be a part of an enzyme substrate complex. It is conjectured that the mast cells play some role both in the early phase of demolition and in the later phase of dedifferentiation to give the cells of the blastemata.

Apart from these possible chemical factors, mechanical factors such as pressure and shearing stresses have been suggested as possible stimuli for dedifferentiation, and Glücksmann (1939) demonstrated experimentally that fibrous tissue could be induced to undergo chondrification under mechanical stresses. It is unlikely that such mechanical factors are the stimulus to this reaction because of their variation in nature and degree from animal to animal. It is more probable that they are concerned with the extent of the volume of tissues formed rather than with the initiation of their process of repair.

Another factor which is known to influence chondrogenesis is the amount of blood supplied to the developing tissue. By using transparent chambers in rabbits' ears, Clark and Clark (1942) showed how cartilage develops only in areas of poor or moderate vascularity. In our study the periosteal blastema differentiated in the absence of any recognisable blood supply until the stage of endochondral ossification was reached, a finding which suggests that vascularity or lack of it may be a controlling factor during healing, but is not the primary factor.

The effect of cortisone on the histochemistry of repair—The first histological evidence of the " cortisone effect " was seen after ninety-six hours and this time lag or delay could be explained by the time taken for the daily injection of cortisone to produce hormonal imbalance with alteration in suprarenal secretions.

There was a reduction in depth of the epiphysial plate due to the disappearance of Phases

II and III—that is, the phases in which the chondroblasts develop into the hypertrophying chondrocytes, with an increase in the intercellular matrix. As in our previous study the early cartilage cells stained metachromatically and used radioactive sulphur. This effect on the "middle zone" of the epiphysial plate has also been produced experimentally by the injection of oestrogens (Gardner and Pfeiffer 1943), which are supposed to act indirectly by inhibiting the secretion of the growth hormone of the anterior pituitary gland (Zondek 1936). Harris (1950) confirmed this work in rats, and related the imbalance of the ratio of growth and sex hormones to the shearing strength of the upper femoral epiphysis and suggested that the imbalance might be a possible etiological factor for the slipping of an upper femoral epiphysis; in rats hypophysectomy causes retarded development of cartilage in this region (Walker *et al.* 1950). In addition to these hormonal factors affecting the cells of the middle zone of the epiphysis, it has been shown by Warren *et al.* (1950) that the cells of the proliferating zone are the most sensitive to radiation from injected ^{32}P. Because of their rapid proliferation and development, these cells are extremely vulnerable to any form of hormonal or vitamin imbalance or even to physical trauma. Therefore care must be exercised in relating histological derangement of this pattern of cells to a specific effect by any particular agent.

In the repair of the fracture the dedifferentiation and metabolism of the cells occurred normally up to the stage of chondrocyte development—that is, the seventh day. This implies that the local stimulus to differentiation is not influenced by cortisone.

After this initial normal phase the chondrocytes appeared to lose their structure and nuclei, and there was a marked increase in the amount of the intercellular amorphous material. This exhibited metachromasia and was radioactive, and appeared to be liberated from the cartilage mass which failed to mature. There was no evidence of endochondral ossification in the abnormal periosteal blastema, although the formation of bone from the medullary blastema appeared to proceed normally. It therefore appears that the differentiation of mesenchymal cells to form osteoblasts and the subsequent intramembranous ossification are not affected by cortisone, its main effect being on endochondral ossification alone.

Layton (1951) described a decrease in the sulphate-fixing capacity after administration of cortisone, and Boström (1953) by *in vivo* experiments showed a small reduction of the ^{35}S content of chondroitin sulphate in a cortisone-treated group as compared with a control group of rats. From the macroautoradiographs in our second series it was seen that ^{35}S was used earlier, and although the two series were not strictly comparable quantitatively because of the different dosages and exposure times, less ^{35}S appeared to have been metabolised. This could probably be related to the decrease in the number of cells using the isotope, rather than to any biochemical effect. Chondroitin sulphuric acid, shown by metachromasia and use of ^{35}S, appeared to be secreted by the differentiating cells up to the critical level of the chondrocyte or mature cartilage cell, whereupon it was liberated as an increased amount of intercellular substance. From the radiographs it appears that calcium phosphate complexes of poor density were being laid down, which suggested an attempt to heal the fracture. However, it is considered that these calcium salts were deposited into the free chondroitin sulphate of the amorphous material. This process would therefore be one of calcification rather than of ossification and, although the radiographs indicated healing, true consolidation by bone formation had not taken place. Attention has been drawn (Urist and Johnson 1943) to the important differences between calcified callus and bony callus which should consist of compact and spongiosa bone. This observation may explain the contradictory reports on the effect of cortisone on the healing of fractures, especially when it has been studied by radiography.

Finally, one other histological point of interest in this series was the formation of mature fibrous tissue across the fracture defect, suggesting that differentiation of the primary mesenchymal cells had followed a different route, although this may not have been a direct result of the administration of cortisone.

SUMMARY AND CONCLUSIONS

1. The utilisation of radioactive sulphur *in vivo* has been demonstrated both macroscopically and microscopically during the preosseous stage of bone repair.

2. The labelled mucopolysaccharide complex, chondroitin sulphuric acid, has been studied during the formation of the medullary and periosteal blastemata in the healing of a fracture.

3. The appearance and possible significance of mast cells adjacent to a fracture, and resulting from the stimulus of trauma, are discussed.

4. Cortisone has been seen to affect the formation of the periosteal cartilaginous blastema and subsequent process of endochondral ossification, with liberation of increased amounts of chondroitin sulphuric acid which was calcified rather than ossified.

The work was carried out while one of us (R. B. D.) was holding the Wilkie Surgical Research Scholarship of the University of Edinburgh.

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SVEN KIÆR

1894-1955

Danish orthopaedic surgery and the Institute for the Care of Cripples in Denmark has suffered a great loss through the death of Sven Kiær, the leader of department II of the Orthopaedic Hospital, Copenhagen.

The sudden death of Sven Kiær on July 20—from coronary occlusion—came as a shock to us all; the day before, he was working as usual at the hospital. In the span of five years we now have lost those three personalities, Guildal, Berntsen and Kiær, who developed orthopaedic surgery on modern lines at the hospital in Copenhagen erected in 1935 by the Society and Home for Cripples in Denmark.

Sven Kiær's contribution to this development was highly significant. He completed his training as a general surgeon and was also for some time consultant in surgery to the child at one of the children's hospitals in Copenhagen before he became an orthopaedic surgeon at the Society and Home for Cripples in 1930. Influenced by his knowledge of the development of orthopaedic surgery in England, he suggested a plan for an orthopaedic hospital in Copenhagen, where fresh injuries should also be admitted, and the actual building of the Orthopaedic Hospital in Copenhagen a few years later in 1935 was based upon this plan. Sven Kiær and Aage Berntsen became assistants to Guildal at this new hospital until Guildal resigned from clinical and operation activities and took over the leadership of the hospital's medical-social functions in 1947, and Sven Kiær was appointed chief surgeon to department II.

Sven Kiær's thorough training and his interests in many different fields of surgery—enlarged by studies in France, England and America—coupled with his administrative, technical and scientific abilities and his remarkable executive power constituted the sound background for the important contribution this gifted personality gave in establishing the recognised standing of the Orthopaedic Hospital in Copenhagen. From his study in France during his training as a young surgeon he brought back his interest in surgery of the sympathetic nervous system. He developed later at the Orthopaedic Hospital as the first of its kind in Denmark an efficient laboratory for the investigation of the function of the vessels and continued to his death to be interested practically and scientifically in the field of vascular diseases and sequels of traumatic injuries. In England he studied plastic surgery and was the first to introduce this field of surgery on a larger scale in Denmark. His work on the refinement and standardising of prostheses and on the treatment of scolioses was also of importance in raising the efficiency of the hospital, and Sven Kiær was one of the first surgeons to introduce acrylic materials in bone and joint surgery.

As a person Sven Kiær was charming. He was loved by his assistants for the comradeship he gave them and for the friendly and happy atmosphere ruling in his department; also for the inspiring example he was to them: Sven Kiær was full of ideas and had the energy to work many of them through to a final conclusion. He was a keen yachtsman, and his summer holidays were often spent on long cruises in Scandinavian waters. Sven Kiær was not only highly esteemed as an orthopaedic surgeon among colleagues in Scandinavia but had many friends among them. Orthopaedic surgeons in other countries also became his friends. We had the good fortune to see many of them from all over the world when Sven Kiær invited them to see the hospital on their way through Copenhagen to the international orthopaedic meeting in Stockholm in 1951. He then organised a "fine show"—a phrase used by one of the distinguished visitors—of the hospital's makings and doings. He repeated this demonstration on a larger scale last year when the Scandinavian Orthopaedic Association had its meeting in Copenhagen under the presidency of Sven Kiær. He was then the natural centre of the scientific discussion as well as of the social gatherings and it was because of him first and foremost that this meeting was as successful as it was.

Sven Kiær's ideas on the development of orthopaedic surgery in Denmark were based upon a broad outlook. It is to be hoped that this outlook will be sustained in the future and that we can continue the contacts with other countries Sven Kiær so happily brought about. But no doubt it will be difficult to be without him. We will remember him as an inspiring personality and a fine and charming colleague and friend. J. M.

J. ALBERT KEY

1890-1955

By the death of J. Albert Key orthopaedic surgery has lost one of its most vigorous intellects and one of its most notable personalities. It is a loss which will be felt especially by the members of the American Orthopaedic Association because to them it will be almost as a personal bereavement. The universal affection for " Albert " was of a kind not commonly encountered when professional men discuss their colleagues.

Not only was he a vital force at every orthopaedic meeting—and he rarely lost an opportuinity of contributing to a discussion or of delivering a paper—but he was also the most colourful character at these meetings. Mention of the name of J. Albert Key evokes a picture of the short, stocky figure stepping aggressively to the rostrum with the audience wondering whether or not he would be serious—but knowing it would be good either way; then the vibrant, humorous voice which needed no microphone, the shrewd, wise comment followed invariably by a sally which always brought the roof down. His one-hundred-per-cent American appearance and manner was a pure delight; and to see him use a target revolver made one accept the legend, probably apocryphal, that he was the son of a " two-gun " sheriff!

But these amiable attributes would be as nothing were it not for the stupendous industry and the astonishing volume of scientific work which came from his pen, either alone or with his collaborators in the Washington University Medical School of St Louis. It was through these writings that he was best known to British orthopaedic surgeons of whom few had the opportunity and privilege of knowing him personally, because by a stroke of ill-luck he was prevented from coming to the Orthopaedic Congress of the English-speaking countries held in London in 1952. Of the few British surgeons who can really claim to have known him there is one who cherishes the memory of an unforgettable week-end spent with him at " Keyhole," his house in the Osarks, in the entertaining company of Mrs Key.

Throughout his career in addition to a full life as a clinician and teacher, his insatiable curiosity made him an inveterate experimentalist, his earliest animal work being done as an interne with Osgood in Boston about 1920. The fundamental problem of osseous union was a constant challenge in his researches; though the whole field of his interests was extraordinarily wide and soon after the recent world war included experimental work into the etiology of disc protrusion.

It would be difficult to imagine a person whose scientific honesty could exceed that of Albert Key. He detested anything that was not genuine and he even went so far in his Presidential address to the American Orthopaedic Association in 1947 to say that he preferred a good general surgeon, drawn to orthopaedic surgery later in life with no special training but just because bones interested him, to a young man trained in a narrow channel of specialisation before he had time to reveal any real aptitude or sense of vocation. To this debunking spirit, even if the idea might not be directly capable of being put into practice, the American Orthopaedic Association, as well as the whole science of orthopaedics, owes a permanent debt.

J. C.

PROCEEDINGS AND REPORTS OF UNIVERSITIES COLLEGES, COUNCILS AND ASSOCIATIONS

GREAT BRITAIN

BRITISH ORTHOPAEDIC ASSOCIATION

The autumn meeting of the British Orthopaedic Association was held in Liverpool on October 6–8, the president, Professor Bryan McFarland, in the chair.

The first day of the meeting was occupied with the Instructional Course, the speakers being *Mr John Charnley* (Manchester) " Disc surgery—the orthopaedic approach," *Mr A. L. Eyre-Brook* (Bristol) " Malignant tumours of bone," *Mr A. M. Hendry* (Birmingham) " Reconstructive surgery in the upper extremity," and *Mr S. A. S. Malkin* (Nottingham) on " Posture."

PRESIDENTIAL ADDRESS

Professor Bryan McFarland, president of the British Orthopaedic Association, gave his address on October 7, 1955. The president said:

I do not know which is the more appropriate quotation on this occasion, whether to say now that the debate is over:

" The tumult and the shouting dies,
The captains and the kings depart,
Still stands Thine ancient sacrifice,
An humble and a contrite heart."

or whether:

" The time has come, the Walrus said,
To talk of many things:
Of shoes—and ships—and sealing-wax—
Of cabbages—and kings—"

Probably both are about equally appropriate. At any rate instead of obeying the Walrus (no reflection on Nobby Clarke) I will try and interest you for a little time in my impression of the changes in orthopaedic surgery during the last thirty years.

In the week beginning September 13, 1925, the operating lists of the three hospitals in Liverpool then doing orthopaedic surgery included the following operations:

Meniscectomy,
Arthrodesis of the foot for paralytic equino-varus,
Several supracondylar osteotomies of the femur for rachitic genu valgum,
Suture of the ulnar nerve,
Arthrodesis of the hip for osteoarthritis,
Several osteoclases of the tibia for rachitic genu varum,
Innumerable " wrench and plaster " for congenital club foot,
Excision of a burns scar and a flap graft on the hand,
Manipulative correction for flexion deformity of the knee in rheumatoid arthritis,
Sequestrectomy of the femur for old osteomyelitis,
Sequestrectomy of the tibia for old osteomyelitis,
Tendon transplant for musculo-spiral paralysis.
Aspiration of tuberculous abscess of the hip,
Multiple subcutaneous fasciotomy for Dupuytren's contracture,
Open stretching of the sciatic nerve for sciatica,
Plaster jacket for backache,
Closed reduction for congenital dislocation of the hip,
Refracture and correction of Pott's fracture,
Refracture and setting of Colles's fracture.

In 1939, just before the second world war had really started, the lists contained again:

Meniscectomies,
Arthrodesis of the foot now known as a stabilisation,
One supracondylar femoral osteotomy for genu valgum,

A displacement osteotomy for osteoarthritis of the hip,
A wedge tarsectomy for old congenital club foot,
A cross leg skin graft for replacement of a scar resulting from osteomyelitis of the tibia.
Sequestrectomy of the tibia,
Extra-articular arthrodesis of a tuberculous hip,
Arthrodesis of the knee for rheumatoid arthritis,
Epidural injection and manipulation for sciatica,
Anterior transposition of ulnar nerve for late ulnar palsy,
Closed reduction for congenital dislocation of the hip,
A bone graft for malunion of the tibia,
Amputation of index finger for laceration of flexor tendons.

In September 1955 the lists were:—
Meniscectomy,
Arthroplasty of the hip,
Stabilisation of the foot,
Open soft-tissue operation for congenital club foot,
Arthrotomy of tuberculous hip and insertion of streptomycin,
Injection of hydrocortone into a knee for rheumatoid arthritis,
Removal of the herniated portion of an intervertebral disc,
Decompression of the median nerve at the wrist,
Transplant of the tibialis posterior and peroneus longus into the calcaneum for commencing paralytic calcaneus,
Fusion of the proximal interphalangeal joints of the toes for claw foot,
Complete fasciectomy for Dupuytren's contracture,
Repair of flexor tendons of index finger,
Application of an abduction splint for congenital dislocation of the hip in a child of seven months.

At first hearing this may seem a rather boring recital of operations, but on closer inspection it is possible to detect a trend and to deduce certain facts. Although meniscectomy remains in its place, arthrodesis of the hip yielded first of all to osteotomy, and then, dissatisfied with providing stability only without movement, or even at its expense, surgeons attempted to secure both by reviving the operation of arthroplasty which had fallen from grace some forty years previously. If we had the full details of the operating lists we would see that big operations on the hips are now performed on patients of an age which would have been considered too great thirty years ago, and we would detect also a tendency to operate more readily on very young children. In other words what was considered the suitable age limit has been both lowered and raised. The lowering of the limit is especially noticeable for instance in operations on children's feet. The almost interminable period of moulding club feet has been shortened; the neglected or intractable foot is much rarer, and operations for late wedge tarsectomy and arthrodesis diminished in frequency. Aspiration of tuberculous abscesses fell away, and then arthrodesis has been replaced by very early opening and injection of streptomycin. That awful open stretching of the sciatic nerve for sciatica, blind and empirical and unsuccessful, followed later by epidural injection and manipulation, equally blind and almost equally uncertain, have given way, because of the knowledge of the condition, to removal of the disc-nucleus which is pressing on the sciatic root. Supracondylar femoral osteotomy and tibial osteoclasis were diminishing in 1939 and had ceased by 1955, because rickets and rachitic deformities were on their way out in 1939 and have now and for many years practically disappeared. Stabilisation of the foot for paralytic deformities has become less necessary because of the balancing of muscle at an early age. The dorsal wedge for claw foot has largely yielded to fusion of the proximal interphalangeal joints. All the various treatments for Dupuytren's contracture have to a considerable extent given way to excision of the dying fibrous tissue, and by using cortisone or something of the sort it is hoped to preserve joints affected by rheumatoid arthritis. Amputations of crushed and lacerated fingers with tendon damage have been superseded by careful repair of the tendons. The management of congenital dislocation of the hip, having gone through a phase in which closed reduction was often carried to an over-optimistic stage, and then through a phase in which a similar attitude influenced open operations, is now concentrating on commencing fairly simple treatment very early.

By 1939 the standards set up for the treatment of fractures and the organisation to deal with these fractures had begun to bear fruit (I won't say whose work this is) and was reaching maturity before 1955. By this year also sequestrectomy and excision of gross scarring from osteomyelitis

have disappeared from the lists because antibiotics have caused more change in acute pyogenic arthritis and osteomyelitis than they have in any other part of orthopaedic surgery.

There are one or two other trends which are perhaps not quite so satisfactory. The number of operations on peripheral nerves have diminished, not because they are no longer necessary but because they are being performed in special centres. The operations of a plastic nature, removal of scars and correction of syndactylism, are disappearing from the lists, again not because they are unnecessary, but because they are being done by other surgeons.

An even greater change is observable in out-patients. No longer, largely speaking, is the pressure in out-patients anything like what it was thirty years ago, when vast numbers of people were being dealt with for the moment by an over-worked out-patient department. Only the most urgent and critical patients were being admitted; repeatedly decisions had to be taken whether it was safe to let the child go home or not: that pressure has largely disappeared. To some extent this is due, as has been indicated, to the crippling conditions themselves having disappeared. It would be fair to give credit for the diminution in disease and the reduction of crippling to the various clinics organised and established by local authorities, *i.e.*, child welfare clinics and orthopaedic after-care clinics; and it might be as well at this point to remember that the credit for promoting this great undertaking ought to go to Girdlestone, Robert Jones, Agnes Hunt and Margaret Beavan. It is obvious also that the easing of pressure in out-patients partly results from spreading the load over an increasing number of out-patient hospital clinics. The increase in such orthopaedic units is very marked. In 1925 three hospitals in Liverpool were doing orthopaedic surgery. There are now fifteen. In the country generally the increase is much greater and probably there are twenty orthopaedic hospital units for every one that existed thirty years ago. This increase has not altogether brought about a drop in the number of people attending an average out-patient clinic because what one might call the character or nature of the out-patients has changed. Thirty years ago the proportion of patients with a definite lesion was something like 75 per cent. To-day I doubt if it is as much as 25 per cent. And of the patients with a definite lesion in those early days many were beyond the point where preventive or conservative measures could be employed, and operation offered the best chance of maximal function; so that about 80 per cent were treated operatively. To-day I should think that this proportion has dropped to something like 50 per cent; in other words only about 12 per cent of all the patients coming to the clinics need operation. This fall in the amount of work, and the pressure in out-patients, has had one very good effect in that it has given time for much better recording of symptoms and signs and conditions, and time for visual or illustrative recording by photographic and other means. It has also allowed the surgeons to think about their patients more, and it has also made it possible for a much better follow-up system to be instituted with increased possibilities for assessing later the relative value of different procedures. This is of course a type of clinical research which is of great importance.

The great changes that have occurred—admittedly gradually and almost imperceptibly—in operations and in out-patient work, are not reflected in the discussions which were held by this Association. The subjects for the 1920s might well be on our programme to-day:

Fractures in the region of the ankle joint,
Internal derangement of the knee joint,
Perthes' disease,
Spastic paralysis,
Injuries of the carpus,
Fractures involving the knee joint,
Fractures in the lower tibial epiphysis (president),
Primary nerve lesions in injuries to the wrist and elbow (past-president),
Fractures and bone-setting (R. J.),
Treatment of fracture of the neck of the femur,
The early treatment of congenital dislocation of the hip,
Reconstructive surgery in paralytic deformities of the leg.

The same is true in the 1930s:

Congenital high shoulder and arthrogryposis,
Fracture of the carpal scaphoid,
Principles of joint mechanics,
Nephrolithiasis in recumbency,
Arthrodesis of the hip,
Osteochondritis of the adult tarsal navicular,
Fracture of the head and neck of the radius,
Tuberculous arthritis of the sacro-iliac, shoulder and knee joints,
Colles's fracture.

In the 1940s of course there was the cloud of war, which threw its shadow very heavily not only on our meetings in themselves, but also on the subjects we discussed. Since 1950 there has happily been a reversion to what one might call peacetime orthopaedics, and a few new subjects appear:

Water content of the nucleus pulposus,
Episodisity in the lumbar disc lesion syndrome,
Surgical treatment of Dupuytren's contracture,
Primary internal fixation of compound fractures,
Osteoid osteoma,
Acrylic prosthesis in arthroplasty of the hip,
Excision of fractured head of radius and acrylic substitution,
Operative treatment of Pott's paraplegia,
Neurectomy for metatarsalgia,
Compression of median nerve in carpal injuries.

In comparing the actual discussions it is noticeable that twenty to thirty years ago there was a much more critical attitude on the part of the audience, though perhaps less on the part of the reader of the paper himself. There is now, I think, generally a very high level of surgical honesty and self criticism. But there is much less dispute and argument: I do hope that this can be revived, and it has been in this hope that I have arranged the two debates and the panel: that the latter was a flop or nearly so was largely due to factors which could be eliminated. At any rate I would like to see free and possibly bitter discussion, even to the pitch of those ding-dong battles between, for instance, William Trethowan and T. P. McMurray, that we witnessed in the 1920s. Perhaps also the chairman might be allowed a little gentle sarcasm such as that indulged in by Sir Robert Jones when a rather dull speaker on a very dull subject kept pausing each time a vehicle passed the building, and Sir Robert, sensing the utter boredom of everybody else as well as himself, in his most innocent and charming manner—and how charming that was—asked the speaker if he should have the traffic stopped. One may also recall his celebrated comment in a discussion of open reduction of fractured tibiae, when he said that this was obviously a most important and illuminating paper: he was sure that whenever necessary this was the method to use: in his experience he had never found it necessary and he doubted if he ever would. However that is just a digression. I was saying that I hoped free discussion can be maintained. Nothing seems to me more futile than for someone to stand and make *ex cathedra* statements which many of his listeners are too polite to dispute. I was also about to point out that compared with thirty to forty years ago it is apparent that to-day more is expected as the result of treatment; in other words the standard of objective has risen. In the first twenty years or so of this century the degree of crippling in the average patient was so severe that to secure a 50 per cent improvement of function was success indeed. The target, if one might so express the degree of function which is aimed at, has steadily gone higher until now quite rightly the target is perfection. I cannot help feeling that we are beginning to realise that this is not always attainable and it is necessary to learn to discriminate between those patients in whom it is possible to reach this target and those in whom it is impossible. This of course is a matter of judgment. But it is something we should try and acquire and then we might arrange when operating to have a second string to our bow which we may bring into use when appropriate.

Now that the pressure of relieving serious crippling has so much diminished, we would be failing in our duty if we did not review the reasons for this and assess the value of the procedures that we have relied on. It also behoves us to promote research on a scientific basis as well as a clinical, so that if possible we may understand more of the nature of the conditions we are treating and the cause of the lesions that have arisen, and in this way may rationalise and modify our operative and other procedures. It is also necessary to try new operations or different forms of old operations. And perhaps I may be excused at this moment if I make a little personal statement on the question of experimental surgery. An editorial which I was induced to write on "Experimental Surgery" may have been construed, in fact seems to have been construed in some quarters, as an indication that I am opposed to experimental surgery. This is very far from the truth. On innumerable occasions I have myself indulged in operations of an experimental nature. It is true that I have waited a fair time before deciding whether they were good or bad or indifferent, but I am not opposed to experiment, and there is no suggestion in what I have written that I am opposed to publication of these experimental procedures. All I say is this, that they should be labelled as experimental and not regarded as great new advances until they have stood the test of time and public opinion.

It is to be deprecated that just now when there is more time to observe, record and consider our work, so much of our time has to be spent serving on committees. It is of course advisable

and right and proper that the medical men in a hospital should play their part in controlling and governing that hospital and directing its policy. It would be a bad thing, incidentally, if the doctors themselves ran the hospitals; the presence and assistance of lay people is absolutely essential. But unfortunately in order to serve the interests of a hospital or a speciality, innumerable reduplicated committees have to be attended. The hospitals are in fact over-committeed and consequently I am afraid they are underdeveloped. Undoubtedly there would be great financial economy, much more development, and very considerable improvement in the use of medical time if control were decentralised and each hospital within an easily arranged financial limit were to conduct its own affairs independently and to follow its own path to the best of the ability of those who were working in it. The machinery of hospital administration is sluggish and clumsy. Unless it is rendered more lively and resilient the hospitals will become bogged down. It is as I have said essential that research be facilitated and encouraged. This country is quite possibly falling behind in progressive and important investigations because of the lack of young, or not so young, medical men who are willing and qualified to devote themselves to research. In order to be useful in research of an orthopaedic nature a young man must have reached the beginning of his senior registrar period and he must be prepared to spend two years on his research work if it is to be fruitful. University fellowships usually carry a salary less than half that of a senior registrar. It is unreasonable to expect our young man to take his foot off the ladder of promotion. It is also unreasonable to expect him to live for two years on a salary which is less than half what he has been receiving, especially as he is probably married and has the responsibilities that implies. It is therefore necessary for the governors of our fate—that is, I presume, the Minister and his advisers—to recognise that a man undertaking research, elected to a recognised research post, should retain the status he has reached within the National Health Service and should continue to be paid the appropriate salary. There are probably other ways of attaining the same objective, but we must certainly do something to promote medical research if we are to keep our place abreast of other nations. I think we have lost the opportunity for being out in front, and I think we can keep our place only if proper impetus is given and proper provisions are made.

And now, having ventured into suggestions for the future, it is perhaps reasonable for a minute or two to consider what that future may hold: how far is orthopaedic surgery going to remain a broad and interesting and progressive subject, or how far is it going to be whittled down until the orthopaedic surgeon is merely a fracture setter, a maker-good of traumatic lesions, and a sorter of pain clinics? I think the answer lies in the hands of the individual orthopaedic surgeon. It is useless to complain about the inroads and encroachments and the intrusions of special specialists in other branches of surgery. It is useless to ask that some authority shall intervene and define the limits of this, that, and the other speciality. The answer to the challenge is for the orthopaedic surgeon to do the work which is properly within his province better than the intruders do it. This raises the question whether orthopaedic surgeons should specialise within their speciality. I must say my feeling is that that is one of the trends that may emerge. Where there is a single orthopaedic surgeon working only on a general hospital (or hospitals) out of contact with orthopaedic colleagues, doing everything—orthopaedic (or near-orthopaedic)—that turns up, it may be that he will lose a good deal of work to his more specialised competitors. On the other hand I do not believe that it is a good thing entirely to segregate orthopaedics and to congregate all the patients into a special hospital, in which the orthopaedic surgeons work in splendid isolation from their general colleagues. Nor do I think that great clinics should be set up on the continental style, with a chef-de-clinique who is virtually a dictator surrounded by subordinate and satellite assistants. I think that the orthopaedic surgeon should continue to work in general hospitals and to conduct open out-patients and accident services in the proper way. In that general hospital he would exercise complete and independent responsibility for everything of an orthopaedic nature which comes to that hospital. But in addition he should hold a second post, and that in a hospital entirely devoted to orthopaedic surgery, staffed by all the other orthopaedic surgeons of the area. In this hospital—or it could of course be an orthopaedic block in a hospital centre—each surgeon would take some special line such as the plastic surgery of orthopaedics or the neurosurgery of orthopaedics, or the surgery of some special part such as the hand, hip or the spine. That I think is a reasonable way in which to arrange to discharge our responsibilities in the future, and that is a way in which we can keep orthopaedics progressive. Because we must progress, we cannot stand still; immobility is the beginning of stagnation and stagnation is the beginning of death. No one can plan or foretell for sure the way in which our actual surgery may advance. We may guess that tuberculosis will go the way of rickets, that cancer will be controlled and that rheumatoid conditions will be eradicated; and we can envisage a great development in the restoration or replacement of lost or useless tissues, and for my part I hope this will be by materials compatible with living tissue and not by using inert materials, which I believe to have a very short future and

are likely to lead us into a blind alley. It is in the discovery of suitable material for tissue replacement that research might immediately be most rewarding.

Although, as I have said, we may envisage these more immediate changes in our art and craft, we cannot possibly foresee long-term advances. Nevertheless we can plan the conditions under which those advances may be made most surely. I believe our plan should ensure that in every possible way research, both clinical and purely scientific, will be facilitated and fostered. It should ensure that research workers will be accorded a status equal to that of clinicians. I say equal because we must take care at the same time that we do not become slaves to science. The clinician, the expert craftsman, will always be essential to the patient. The plan should also ensure that in addition to dealing with general orthopaedic work particular interests and special skill will be developed and encouraged. It is for us to have a plan and to see it through, just as it is our duty to preserve and enhance the great heritage which was handed down to us thirty-odd years ago.

DEBATE

On the morning of October 7 a debate was held on the motion "That Lucas-Championnière was right."

Professor George Perkins (London) opened for the motion with characteristic humour. Lucas-Championnière, he said, had written a book on fractures, the basic theme of which might be summarised as " to hell with splints." While the speaker could not support this *in toto* the idea was certainly more right than wrong. In discussing the harm done by splints he said that splintage caused stiffness and this did not always disappear with time. Stiffness resulted from 1) inextensibility of muscle; 2) incongruity of joint surfaces; and 3) organisation of exudate. If a muscle was held in the shortened position it lost its ability to extend and muscle tone was inhibited. The only way to prevent this loss of extensibility was to stretch it occasionally to the full, but splintage made this impossible. With regard to the joint it was impossible to reproduce a perfect surface with most fractures, but early movement did mould the joint surfaces and produce congruity. At the same time avoidance of splintage prevented the muscles binding together from the organisation of haematoma and exudate.

In considering the good a splint might do, he separated splintage into two types, the *pure*, which was prolonged, uninterrupted, rigid and extensive, and the *simple*, which was none of these things and was simply used to maintain reduction when displacement would otherwise recur. The " splinters," he said, held the tenet that because a few need it, all need it. Only three fractures needed " pure " splinting, those of the neck of the femur, the scaphoid, and the mid-shaft of the radius, the latter because no other method would control rotation strain. Hinging movements and rotation were the causes of fibrous union, because they stretched the early callus. The philosophy of the " non-splinters " lay in their faith that with few exceptions muscles alone would prevent this stretching. There was no need to set a fracture if there was no displacement, if the displacement did not matter, or if reduction was impossible. Finally he proposed that the non-splinters' creed should be: 1) don't burden the patient with purposeless splints; 2) a " simple " splint does less harm than a " pure " splint; 3) only a few fractures need " pure " splintage; and 4) reduce the use of " simple " splintage to the minimum.

Sir Reginald Watson-Jones (London), opposing the motion, said that Lucas-Championnière (1843–1913) was a pioneer; splintage in his era was very complete and extensive, and he did not know that it was possible to hold bone still and yet continue to use the overlying muscles. Lucas-Championnière had said that perfect reduction was neither possible nor desirable and had advocated massage and mobilisation of *all* fractures. Sir Reginald could not accept the opener's differentiation of types of splintage; if one fracture needed immobilisation, they all needed it. He believed that if a fracture was to unite with certainty it must be immobilised completely, and all fractures would unite if they were immobilised for long enough. Non-union resulted from splintage that physiologically was not complete enough, or not maintained for long enough. We should not fear the *temporary* resultant stiffness; if functional activity was maintained, there was rapid recovery of a full range of movement. The immobilised knee or shoulder always recovered its movements if reliance was placed on active exercises alone. Many new hip operations were based on the fundamental, but in his view unfounded, fear of permanent knee stiffness if this joint were immobilised, but in 200 cases of hip arthrodesis with the limb in plaster for many months less than 8 per cent had stiff knees. All the fears of Lucas-Championnière would have been put at rest had he had our knowledge of the need for maintaining muscle activity in a splinted limb.

Mr Norman Roberts (Liverpool), seconding for the motion, maintained that what Lucas-Championnière had really said was that the immobilisation of fractures as practised in his day

gave pride of place to union and disregarded function; the resultant withered limbs were worse than the results of no treatment at all. Böhler and Watson-Jones had recognised the dangers and assured us that with their methods of treatment the danger was over; the trouble was that we had believed them! The speaker thought that immobilisation was losing ground year by year. There were only two indications for splintage; to make a fracture unite which would not otherwise do so, and to keep a fracture in good position. He spoke cogently on the perils of internal splintage and said that nothing but good could follow the abandonment of unnecessary splintage, especially in joint fractures. According to the speaker, who demonstrated his possession of the book, or at least of a book, by Lucas-Championnière, the latter had not said that his method was applicable to all fractures—he certainly immobilised some fractures of the tibia and fibula. In conclusion Mr Roberts remarked that injured shoulders *do* stiffen—with or without immobilisation.

Mr Robert Roaf (Oswestry), seconding for the opposition, advised the meeting to leave the realms of fantasy and get back to reality. He then played a long recording of quotations from Lucas-Championnière, which the president obligingly translated for those without his knowledge of the French language as "Massage is a good thing." While the speaker had the greatest respect for his opponents, he maintained that Mr Roberts was in reality an exponent of perfect reduction and fixation and he was therefore quite unable to believe what he had just said. Lucas-Championnière was an arrogant writer but no true scientist, a believer in "all or nothing," a surgical Karl Marx. Innovations must have a scientific basis; even now we had no fixed concept as to what constituted union and when, but Lucas-Championnière maintained that massage and passive movement reduced the fracture and promoted union, a statement for which he produced no evidence whatever. He looked backwards to the mysticism of the Middle Ages, and he lacked the true artist's "feel" for his material. "In science dogmatism is always wrong." The principle of reducing a fracture and holding it reduced remained a sound one. John Hunter had demonstrated the close connection between form and function, but Lucas-Championnière believed in neither. To vote for Lucas-Championnière, he concluded, was to vote against Hunter.

The motion being then open for discussion, *Mr Philip Wiles* (London) said that the conflict was as old as surgery itself; the bone needed rest, the soft parts needed mobilisation. The advocates of the conflicting beliefs might be compared to fascists who regimented everything and anarchists who preferred a free for all. In reality right lay between the two. He remarked on the benefits to be expected from the use of hyalase in reducing the binding in soft parts, and said he would prefer to vote for a centre party which made use of the best in both creeds. *Dr Rex Diveley* (Kansas City) said he too prefered the middle road; both sides were right. He demonstrated the difficulty of deciding the right course of treatment in an example of bilateral fractured neck of femur. *Mr H. Osmond-Clarke* (London) said that the coming of Sir Reginald Watson-Jones to the London Hospital had resulted in a reduction to vanishing point of fractures needing a bone-graft for non-union, but that fortunately for the training of their registrars this loss was replaced by material from other hospitals where different methods were employed. *Mr G. Fisk* (London) supported the motion. The physiological immobilisation of Watson-Jones was in reality physiological mobility. The contractile effect of the muscle tube gave the earliest recovery of function, and he had had only one case of delayed union (due to muscle interposition) amongst 120 fractured femora treated by early movement. *Mr F. G. Ward* (Staines) was convinced that Lucas-Championnière was right; a stiff limb was often worse than a pseudarthrosis. *Mr D. Wainwright* (Stoke-on-Trent) felt that there was a degenerate ring about "the book," of which he too boasted temporary possession. Written in 1910, it reflected the terminal period of the surgeon's life; his clientele was then aristocratic, and largely feminine with perhaps minor fractures, and he felt it was desirable that the massage and movements should be applied personally. *Mr John Charnley* (Manchester) thought that the middle course, as advocated by Mr Wiles, was dangerous. The motion was either right or wrong; he felt that massage was the real basis of Lucas-Championnière's theme. There were three possible causes of failure to unite: 1) the callus never crossed the gap; 2) the callus succeeded in crossing the gap, but was too fragile, broke down, and reverted to (1); and 3) the callus would cross the gap successfully if only it were immobile. If a bone were plated, only (1) could be relevant because immobility was far greater than could be achieved by any other method. He had ventured a "Law of Conservative Treatment": full mobility could be restored if all bones and joints in a limb were fixed no longer than the physiological maximum of eight weeks. If union were delayed and longer fixation were required, joint stiffness would follow. But prolonged splintage alone was not the cause; there was some other factor. *Mr F. G. St C. Strange* (Canterbury) said that in Nature immobilisation of a fracture was unknown. The principles of reduction, maintenance of reduction, and restoration of function were absolute; but maintenance of reduction and immobilisation had often been confused. We should take a lesson from Nature. He believed that all fractures would unite if *not* immobilised except those where there was-interference with

the local blood supply. *Mr Osmond-Clarke* (London) remarked that in spite of this veterinary surgeons shot horses with broken legs. *Mr W. Sayle-Creer* (Manchester) said that this was merely because it was uneconomic to treat the horse. He found the debate confusing because the terms of reference were too loose. Quoting the instance of the fractured calcaneum, he said that no treatment was only modified treatment. The failures in fractures were due to over-treatment. *Mr A. N. Birkett* (Nottingham) said that immobilisation vitiated Nature's way of stimulating union by muscle spasm, and we needed some method of replacing this stimulus to callus formation during splintage. *Mr C. H. Cullen* (Manchester) related his experiences in treating a fractured femur in a St Bernard dog on cosmetic indications. *Mr M. Alms* (Liverpool) discussed the importance of the patient's state of mind. The hunger stimulus forced the injured animal into activity; there was no such human stimulus in the Welfare State. Ninety per cent of children needed no treatment for their fractures; they were still at the animal stage. The madman with no sense of pain and the tough keen extrovert both did well. He felt that both the openers were right; they sold themselves to their patients by virtue of their enthusiasm. *Professor Walter Mercer* (Edinburgh) called attention to a patient whose ankle and knee were immobilised for two years while an arthrodesis of the latter proceeded to fusion. At the end of this period the ankle was completely stiff, but within two months he had gained normal mobility.

Professor George Perkins (London), in summing up, remarked that the Pyrford hip arthrodesis was designed with the object of putting the hip at rest, not of leaving the knee free. Lucas-Championnière should be accepted as no more than a great figure in medicine and a pioneer of physiotherapy.

Sir Reginald Watson-Jones (London) said that non-union and mal-union *did* matter. He agreed that immobilisation was bad for the soft tissues, but reiterated that he kept the soft parts moving.

The motion was then put to the meeting and defeated by ninety-four votes to seventy-three.

CLINICAL MEETINGS

On the afternoon of October 7 members enjoyed clinical and operative demonstrations at the Royal Infirmary, the David Lewis Northern Hospital, the Royal Southern Hospital, Clatterbridge Hospital, Alder Hey Hospital, and Broadgreen Hospital.

PAPERS

On Saturday, October 8, the new president, *Mr Philip Wiles* (London), took the chair.

Recurrent dislocation of the shoulder—*Mr J. W. Dickson* (London) and *Mr M. B. Devas* (London) had reviewed recurrent dislocation of the shoulder with special reference to the late results of Bankart's operation. Bankart had left no detailed follow-up. They had collected ninety-five operative repairs by Bankart himself or strictly according to his technique. Fifty patients were available for a follow-up made with special reference to the presence of the lesion he described, the range of movement after operation, and the recurrence rate. The initial trauma followed no set pattern, and they had found no association between the side of the dislocation and hand-dominance. The classical lesion was present in thirty-two, absent in nine, and indefinite in nine. Fifty per cent had less than 10 degrees loss of lateral rotation; seven patients had more than 60 degrees loss of lateral rotation but in only three of these was it directly attributable to the Bankart operation itself. Recurrence of the dislocation occurred in two patients, but in each instance was due to severe trauma; neither had a defect in the humeral head. They concluded that the operation was highly effective with no significant resultant loss of movement, and they showed a film of a thirty-year result in the seventh case on which Bankart had operated.

Mr H. Osmond-Clarke (London) said that only two operations regularly enabled a man in the Services to return to A1—the Bankart and the Putti-Platt. He felt that a substantial majority had a defect in the humeral head and any operation that permitted this area to come into contact with the glenoid was liable to produce a "cog-action." In a personal series of over 100 cases, he had had only two recurrences, both readily explicable. Loss of lateral rotation gave rise to no disability. He advised the use of either operation, but said the boring of the necessary holes in the glenoid margin in the Bankart operation was difficult. *Mr A. L. Eyre-Brook* (Bristol) asked the speaker what was done when no Bankart lesion was found. The reply was that the redundant capsule was excised and the margin resutured to the front of the glenoid, a procedure basically like what was done when the lesion was present.

Displacement osteotomy of the femur—*Mr D. Wainwright* (Stoke-on-Trent) had reviewed sixty-nine cases of displacement osteotomy in the treatment of disorders of the hip joint, four

for old congenital dislocation, four for congenital coxa vara, three for slipped epiphysis, seven for fractures of the femoral neck, three for old septic arthritis, twenty-two for tuberculous arthritis, and twenty-six for osteoarthritis. In the fractures there was a fair chance of producing union if the operation was done early enough. In the tuberculous cases ten out of eleven with fibrous ankylosis proceeded to bony fusion. Of eleven with active disease seven fused by bone. In the osteoarthritic cases relief of pain was almost constant, and improvement in the joint itself might continue as long as fifteen years. Five of these, all very severe and stiff before operation, proceeded to bony ankylosis. He remarked that even with inadequate displacement results were often very good, and wondered why this was so and whether the displacement was really necessary. He thought that the provision of a pelvic *point d'appui* was not vital, but that the operation worked by correcting deformity and correcting the lines of stress. It was possible that the mere division of the bone produced some beneficial vascular change. In conclusion he paid tribute to T. P. McMurray and reminded the audience that in his hands the operation used to take five minutes and involved the use of a knife, an osteotome, a pair of forceps, and three short pieces of silk.

This paper promoted a brisk and prolonged discussion. *Professor J. Trueta* (Oxford) discussed the reasons for the apparent thickening of the joint cartilage which followed the operation. The changed mechanics might allow some regeneration. Pauwels said it was the varus which helped, but McMurray had said it was the valgus. In reality it was probably the freeing of the femoral head, so that one was making use of an unworn area of cartilage in the new region of stress. He had noted that patients with the best results walked in slight flexion, and he thought that the results in the tuberculous cases confirmed that an increased blood supply was an important factor. *Professor Bryan McFarland* (Liverpool) said that if the operation was done under local anaesthesia it was apparent that division of the bone itself did cause pain. *Mr K. I. Nissen* (London) thought that the answer lay in Trueta's work, whence it appeared that pain was related to hyperaemia and that it was the diminution of blood supply that was beneficial. Eventually it might prove unnecessary to divide the bone completely; some minor local intervention might be equally successful. *Mr D. Wainwright* (Stoke-on-Trent) felt that the present trend to use internal fixation after the displacement might vitiate the effectiveness of the operation.

Acute osteomyelitis—*Mr C. H. Cullen* and *Mr A. Glass* (Manchester) reviewed 206 cases of acute osteomyelitis followed up for two to five years. The proportion of males to females was 2:1. The tibia was involved in seventy-two, the femur in sixty-five, and the humerus came next in frequency; in fourteen the infection started in the diaphysis. They were treated by antibiotics and splintage with or without operation. Twelve per cent were caused by penicillin-resistant organisms. The majority operated on had a primary suture. A high proportion developed marked bone changes, especially in the upper humerus and lower tibia. There were three deaths in the series, two within twenty-four hours of admission from septicaemia. The recurrence rate was 9·7 per cent, but this was nearly twice as high in patients subjected to operation. Early chemotherapy reduced the bone changes. The decision to operate should be made early, and successful primary closure of the wound reduced the chances of the infection becoming chronic. The chronicity rate was the same whether or not the bone was drilled at the time of operation. They concluded that chemotherapy was no "cure-all"; acute osteomyelitis still remained a serious progressive condition. Chemotherapy must be started early, and a well-timed operation was still helpful. Finally they stressed that this was an orthopaedic problem which could not safely be left in the hands of the paediatricians. *Professor J. Trueta* (Oxford) said that he operated on all cases that retained tenderness after twenty-four hours' chemotherapy and always sutured the wound. *Professor R. I. Harris* (Toronto) said that in Toronto they had found that the use of penicillin together with sulphonamides had eliminated the need for operation, even when sequestra were present, but in Australia, as in Britain, antibiotics alone had not completely controlled the disease. *Mr C. H. Cullen* (Manchester) said that the decision to operate was usually made after the third day, 1) if there was an obvious abscess, 2) if the local signs were not settling, and 3) occasionally if the soft tissue shadows in radiographs indicated the need. They tried to avoid operation; the newly born were capable of resorbing even large sequestra. *Mr R. Roaf* (Oswestry) pointed out that early operation had the advantage of giving immediate information about the sensitivity of the organism. *Professor Bryan McFarland* (Liverpool) said that one great advantage of operation was the consequent relief of pain. *Mr A. Glass* (Manchester) said that latterly a blood culture had been carried out as a routine on admission.

Volkmann's contracture—*Mr H. J. Seddon* (London) emphasised that his personal experience was extensive only because patients had been sent to him from all over the country; in many ischaemia had apparently been inevitable, and the supervision of the fracture treatment had not been the responsibility of members of the British Orthopaedic Association. In the forearm there was an ellipsoid zone of ischaemia, worst at its centre. The deep forearm flexors were also involved,

and the median nerve passed through the centre of the zone. Flexor digitorum profundus and flexor pollicis longus were the most affected. He recognised three grades; those where the changes were reversible, those leading to some fibrosis, and those with severe fibrosis and necrosis. The nerve lesion might be recoverable if there had been no more than Wallerian degeneration distally, but often there was a length of thin, dirty yellow nerve through which axons could never pass. He advised that after a delay of three months the complete infarct, which acted as a foreign body and stimulated fibrosis, should be excised, thus converting the forearm into a condition comparable to that after a severe injury: operation was not always required. The problems of repair were then assessed. Various tendon transplants were feasible, and nerve grafting or repair by nerve pedicle was usually required to bridge the large gap. He thought it better to complete the tendon operations before undertaking repair of the nerve for fear that the former, if done subsequently, might interfere with the recovery of the nerve graft. Assessment of the need for a nerve graft was helped by biopsy at three different levels; a graft was certainly required if the nerve narrowed to less than one-third of its normal diameter.

Treatment of osteochondritis dissecans—*Mr I. S. Smillie* (Dundee) said that it had been known for ten years that healing of the lesion was possible in children, but this potentiality was lost at adolescence. He proposed deliberately to over-simplify the question of etiology and regard it as a fracture which was never seen in the acute stage. Where the fragment remained in situ, he regarded the position as one of delayed union, requiring rest and improvement of local blood supply. He had obtained union and healing by plaster immobilisation alone in three cases, and by drilling and immobilisation in fifteen. When the fragment had shifted from its bed the condition was one of established non-union. He had treated eight such cases by clearing out the bed, reshaping and replacing the loose body, drilling, and fixation by screw or special pins which were removed when union had occurred. The simple drilling operation might need x-ray control if an intact articular cartilage made localisation uncertain. He stressed that it was difficult to fix the loose body in its bed and that this was only a preliminary report. He suggested that it might later be found possible to make use of frozen homografts to fill the cavity, and that this might open up great possibilities in joint surgery generally. *Mr G. Osborne* (Liverpool) showed slides of an example where he had fixed the separated fragment with a peg bone graft.

Cartilage grafting—*Mr K. H. Pridie* (Bristol) talked on cartilage grafting and a new approach to the treatment of osteochondritis dissecans and osteoarthritis of the knee. In osteochondritis dissecans he believed that a vascular calamity set up a chain of events finally leading to osteoarthritis; there was a cold area in a hot joint. As an experimental procedure in a few cases he had tried removing the loose body, excising the infarct, packing the resultant cavity with cancellous bone chips and covering them with a thin graft of articular cartilage taken from the opposite femoral condyle. The knee was then immobilised in extension for three to six weeks. He claimed that the results were satisfactory. He had applied the same principles to the eburnated condyle in osteoarthritic knees, gouging down to healthy bone and covering it with cartilage. He said that the cavity so formed filled up. He also showed films of a knee in which he had drilled the eburnated condyle, but he did not advocate this as a method of treatment.

The articular damage in complete ruptures of the lateral ligaments of the ankle—*Mr J. Rowland Hughes* (Wrexham) pointed out that whereas articular damage resulting from habitual dislocation was an accepted fact, the damage which occurred at the time of injury and subsequently in complete ruptures of the lateral ligaments was perhaps not realised, as it was hardly ever demonstrable except at operation. A series of thirty-four ankles with complete rupture of the lateral ligaments had been explored with a view to primary repair of the ligamentous injury: all presented as recent injuries, but at operation twelve were shown to be old or recurrent lesions. The articular lesions in old and recent cases were illustrated and the findings were summarised as follows: 1) Some articular damage can be found in 50 per cent of all cases, from minor fissures to osteochondral fractures. 2) The incidence of articular damage was virtually the same in recent and recurrent lesions, suggesting that the initial injury was the determining factor in subsequent articular changes. 3) Although the incidence in both groups was the same, the nature of the lesion was modified by recurrent subluxation. 4) The site of articular damage was nearly always constant, being situated on the supero-lateral border of the talus, about 3 centimetres from its anterior extremity.

Problems of internal prostheses—*Dr J. T. Scales* (Stanmore) discussed internal prostheses: the problem in relation to materials and construction. Prostheses might be permanent or temporary, weight-bearing or non-weight-bearing, free or fixed. In design one had to consider the properties of the material, the forces to be withstood, the response of the tissues, and the surgical technique. Many materials were inert if implanted in muscle experimentally. With plastics, resistance to abrasion was low, and thus polymers might respond badly to weight bearing. Abrasion of acrylic

resin prostheses in the hip gave rise to particles probably of colloid size, but they were much bigger from nylon, while with polythene they were even larger. The size and quantity of the particles appeared to influence the type and degree of tissue reaction. Adding 18 per cent of chromium to iron alloys greatly increased their corrosion resistance; nickel and molybdenum helped this further still while improving their mechanical properties. He stressed the importance of the surface finish of prostheses; minute unpolished areas acted as corrosion centres. Furthermore various errors in the manufacturing processes of these crystalline ferrous alloys might stress the "grains" or crystals, and corrosion might originate at the boundaries of such "stressed grains." All implants were liable to fatigue, and the combination of corrosion and fatigue was particularly liable to cause failures in the prostheses, for example cracks in hip cups. The speaker then discussed the electrical effects between dissimilar alloys and pointed out that there was an appreciable transference of metal from the tools employed during surgery to the screws, etc., which had been shown to aggravate this effect. Saw blades, necessarily made of a different type of steel, were potential offenders. There had been no known failure due to corrosion in chrome cobalt alloys. He thought that such elements as titanium and zirconium might have great possibilities, but there was always the factor of what stresses the tissues themselves could withstand. Finally he made a plea for further study of these very difficult problems and for some form of standardisation such as was being developed in the United States.

OFFICERS FOR 1956 57

The following were elected: Vice-president—Mr Andrew Fowler (Aberdeen); Honorary Treasurer—Mr L. W. Plewes (Luton); Members of the Executive Committee—Mr Roland Barnes (Glasgow); Mr John Bastow (Bath); Mr John Charnley (Manchester); and Mr R. C. Murray (Inverness).

ELECTION OF FELLOWS, MEMBERS AND ASSOCIATES

The following were elected Fellows:

F. G. Badger (Birmingham),
David Brown (Sunderland),
T. J. Fairbank (Cambridge),
L. Gillis (London),
G. V. Osborne (Liverpool),
A. R. Parkes (Glasgow),
D. L. Savill (Edinburgh).

The following were elected Members:

C. F. Chapple (Mansfield),
R. H. V. Hafner (London),
T. M. Hennebry (London),
J. P. Jackson (Mansfield),
P. R. N. Kerr (London),
J. B. Kyle (Hexham),
M. D. Leitch (Middlesbrough),
G. C. Lloyd-Roberts (London),
G. P. Mitchell (Edinburgh),
R. D. Muckart (Bridge of Earn),
B. Mukopadhaya (Patna),
Dennis Walker (Ashford),
Garthowen Williams (Nairobi).

The following were elected Associates:

K. L. Barnes (Manchester),
L. Bowcock (Newcastle, Staffs),
R. A. Bremner (Oswestry),
B. H. Brock (Bristol),
J. J. Commerell (Cape Town),
P. I. Cromack (London),
G. E. Dunkerley (Portsmouth),
R. B. Duthie (Edinburgh),
J. C. Foster (Bradford),
P. R. French (London),
A. J. Harrold (London),
J. P. Heron (Liverpool),
J. A. James (Stoke-on-Trent),
H. A. Kidd (London),
M. L. H. Lee (Cardiff),
P. H. MacAuley (Oswestry),
T. J. Mills (Oswestry),
F. H. Moore (Dundee),
T. H. Morgan (Oxford),
C. B. Wynn Parry (Royal Air Force),
R. W. Povey (Alton),
A. E. Pritchard (Liverpool),
J. B. Pyper (Belfast),
W. C. Robinson (London),
B. Sankaran (Madras),
W. Sillar (Glasgow),
A. J. Spence (London),
I. M. Stewart (Bridge of Earn),
K. S. Stewart (Inverness),
H. Preston Watson (Port of Spain),
A. G. M. Watt (Glasgow).

1956 MEETINGS

The Spring meeting has been arranged for May 3–5 at Gleneagles, Scotland. The Autumn meeting will take place about the first week of October in Rome, Florence and Bologna.

ROBERT JONES GOLF CUP

In the most stormy weather that has been encountered since the inauguration of the Robert Jones Golf Cup nineteen orthopaedic surgeons battled their way round the links of the Royal Liverpool Golf Club on Thursday, October 6, 1955. In the midst of many high scores, Mr J. P. Campbell of Nottingham put in the excellent return of 97—15=82. The runner-up was Mr R. H. Maudsley of London who, from a handicap of 8, returned the very laudable score of 83 net. After the competition we were very honoured to be able to have a dinner for golfers of the association in the historic club house of the Royal Liverpool Golf Club and we were delighted to have as principal guest the captain of the Club.

After dinner Mr Gordon Irwin was elected captain of the golf section for the ensuing two years.

The Spring meeting of the Orthopaedic Association in 1956 is being held at Gleneagles Hotel and it is proposed to hold the next Competition for the Robert Jones Cup on Thursday, May 3, 1956, on the King's Course situated in the grounds of the hotel. Further details will be circulated later by the Golf Secretary, Mr J. V. Todd of 20 The Grove, Gosforth, Newcastle upon Tyne, 3.

TRAVELLING FELLOWSHIPS IN ORTHOPAEDIC SURGERY

from Great Britain and the British Commonwealth to the United States of America in 1956

Applications are invited from Fellows, Members and Associates of the British Orthopaedic Association, whether resident in Great Britain, Ireland or elsewhere in the British Commonwealth of Nations, for election as Travelling Fellows to visit orthopaedic centres in the United States of America and Canada for an eight-week tour (including the time of transatlantic travel). Five Travelling Fellows will be appointed by the president of the British Orthopaedic Association on the recommendation of the Joint Committee for Postgraduate Orthopaedic Education. In the election, preference will be given to senior orthopaedic registrars and surgeons who have gained orthopaedic consultant appointments within the last few years. The party will probably leave this country early in May and return at the end of June.

Twenty typewritten copies of the application should reach the secretary of the British Orthopaedic Association, 45 Lincoln's Inn Fields, London, W.C. 2, not later than December 31, 1955, stating name, address, telephone number, age, qualifications, medical school, and present appointments, together with an outline of previous experience and academic or literary achievements. The application must be supported by a referee who is a Fellow of the Association.

If any young surgeon who is not already an Associate of the British Orthopaedic Association wishes to apply, he should communicate with the secretary of the Association at 45 Lincoln's Inn Fields, London, W.C. 2 (HOLborn 3474) who will send the necessary forms.

ROYAL COLLEGE OF SURGEONS OF EDINBURGH

450th ANNIVERSARY 1955

How fortunate we are to have a Monarchy—a gracious Queen and her family. It is small wonder that we are envied throughout the world for this privilege. In every mind there is an ideal; in every heart there is need for someone to represent that ideal—someone to look up to with respect, affection and loyalty, someone who will befriend and defend the rights of an individual against every tyranny. These were the thoughts that passed through my mind as I listened to His Royal Highness, Prince Philip, Duke of Edinburgh, speaking to the Fellows of the Royal College of Surgeons of Edinburgh. It was the evening of Monday, June 20, 1955. The College was celebrating the 450th Anniversary of its Charter Day. To mark the occasion His Royal Highness, Patron of the College, had graciously consented to receive its Honorary Fellowship and afterwards to dine with the Fellows. The ceremony of enrolment was conducted in the Hall of the College. The Very Reverend Charles Warr, Dean of the Thistle and Chapel Royal, constituted the session by reading the College prayer, traditionally ascribed to John Knox who so long ago was a predecessor of Dr Warr as Minister of St Giles' Cathedral. The senior Vice-President, Professor R. C. Alexander, presented His Royal Highness for admission to the Honorary Fellowship in an address which deeply moved the Fellows seated at the dining tables round the Hall. Never in its 450 years of existence had the College added to its Roll of Honour one more worthy than the Duke, who had brought to his arduous duties an enquiring and a discriminating mind, a keen interest in scientific progress and a concern for the welfare of his Queen's subjects. His qualities of leadership, friendliness and understanding were recognised clearly and valued highly. By skilful surgery the " top table " had been dissected; the centre part had been removed and by prosthetic replacement a baize-covered section was substituted. On this lay the Roll Book. Having taken the College oath His Royal Highness signed his name. He was

formally enrolled by the president, Professor Walter Mercer. The prosthesis was removed and the Duke passed into the ranks of the Fellows; the dining table was restored to normal and at the appropriate moment His Royal Highness proposed the toast of the Craft of Surgery. As Patron of the College he gave himself a hearty welcome as the youngest Fellow. He said that no doubt science was wonderful but if ever again he came under the knife and anyone had "to tinker about with his insides," he would prefer an accomplished craftsman rather than an expert experimental scientist. It need hardly be added that this was received by the Fellows with prolonged applause.

The president's reply was a joy to hear. In this, as in all he did that evening, his touch was perfect and was an index of his demeanour throughout the whole of the celebrations. He was dignified, gravely courteous, and yet warm and friendly, combining a proper regard for tradition with a merry twinkle in his eye.

John Bruce, vice-president of the College, proposed the toast of the guests in a speech which might well serve as a model on any such important occasion. As might be expected the response by the Right Honourable David Viscount Kilmuir, Lord High Chancellor of Great Britain, was a model of keen perception, brilliant ideas and happy phrases.

His Royal Highness, the Duke of Edinburgh, was kind enough to accept as a memento of the occasion a silver replica of a bowl used for "blood-letting." Symbolising the great age of the craft of surgery, in which though in a different way the procedure still has a place, the bowl formed a most appropriate present. With "*Floreat Res Medica*" the transactions and entertainment of the evening were over. It was felt that the Royal Charter had been renewed.

On Tuesday, June 21, the Fellows of the College, the Honorary Entrants, distinguished guests and interested friends attended at St Giles' Cathedral a service of thanksgiving and commemoration conducted by the Rev. Dr H. C. Whitley. In the mellow atmosphere of this ancient building with its old walls, speaking history, and with its rich stained glass glowing with beauty, the service was memorable for tranquillity, faith and gentleness. The present Minister of St Giles', Dr Charles Warr, the College Chaplain, gave the address. He dealt particularly with Christianity as the fundamental basis of Medicine and his thoughtful words will not be forgotten. Many of his listeners must have rejoiced in their own dedication of the quiet art of healing.

From the Cathedral the company moved to the University, where luncheon was provided in the Great Hall, the colour and decorations of which may be austere but with such proportion and architecture as to give an impression of restrained magnificence. In such a setting it was natural that the proceedings should be dignified. Sir Harry Platt struck exactly the right note when he proposed the Royal College of Surgeons of Edinburgh in a speech which was enlivened by humour. In reply Professor William Johnstone went back in his memory fifty years to the 400th Anniversary which he had attended as a representative of senior students and junior surgeons. Unfortunately "that old bald cheater Time" robbed the president of a full opportunity of toasting the guests, robbed Sir Edward Appleton of his reply, and robbed us all of the pleasure of hearing all they would have said.

The hour came for conferment of the Honorary Fellowships in the Hall of the College of Surgeons—a room remarkable for its stateliness and charm, large enough to be impressive, but small enough to create an atmosphere of friendship. The chaplain constituted the meeting by reading the College prayer. The president referred briefly to the history of the College.

The Right Honourable the Viscount Kilmuir, P.C., G.C.V.O., Lord High Chancellor of Great Britain, was presented for admission by David Skene Middleton whose address carried words of distinction and was delivered in a tone which conjured something of the magic and mystery of Scotland. It almost brought to life the portraits round the Hall. Had their subjects suddenly stepped out of the frames and marched up and down to the plaintive note of the pipes none would have been surprised.

His Grace the Duke of Hamilton and Brandon, P.C., K.T., G.C.V.O., LL.D., a remarkable pioneer of aviation, was presented for admission to the Honorary Fellowship of the College by James Sneddon Jeffrey.

The Right Honourable the Earl of Rosebery, P.C., K.T., D.S.O., M.C., was presented for admission by Thomas McWalter Miller who made it quite clear how great was this representative of a great family.

The Right Honourable William Shepherd Morrison, P.C., Q.C., M.P., Speaker of the House of Commons, was presented *in absentia* by Douglas Miller who gave a close and wistful picture of a man so universally respected.

Sir Edward Appleton, G.B.E., K.C.B., F.R.S., LL.D., was presented for the Honorary Fellowship of the College by Sir James Rognvald Learmonth whose presentation was characteristically brilliant.

The Very Reverend Charles Laing Warr, K.C.V.O., D.D., LL.D., R.S.A., F.R.S.E., was

To

The Royal College of Surgeons of Edinburgh

from the

Journal of Bone and Joint Surgery

The Journal of Bone and Joint Surgery in its representation of the English-speaking world has special privilege in offering congratulations to the Royal College of Surgeons of Edinburgh on its four hundred and fiftieth Anniversary for the reason that the President of the College is also Chairman of the British Editorial Board

Every Surgeon of the English-speaking world offers congratulations and pays great respect to the history, traditions and accomplishments of this great Royal College

Reginald Watson-Jones

British Editor

June 1955

presented for admission by Keith Paterson Brown whose words revived the feeling of reverence engendered by the morning's service.

Dr William Edward Gallie, M.D., LL.D., D.Sc., F.R.C.S., was presented for admission by Bryan McFarland who said: " William Edward Gallie has dedicated his heart and soul to the study of medicine, and devoted his head and hands to its practice. Already, in his own country he has achieved greatness, and has been acclaimed throughout the world as a leader of surgeons. It is doubtful whether a man can receive greater recognition than that his fellows shall treasure and acknowledge his wisdom, skill and humanity in the way that the medical men of Toronto, and indeed of all Canada, pay homage to Edward Gallie. He was born in Barrie, Ontario, sixty miles north of Toronto. It seemed likely that he might become a lumber merchant or builder, but he found that shingling a roof was such monotonous drudgery that he determined to earn his living by more interesting and easier work, and so he entered Medicine—and ever since has worked far harder than any builder ever worked. What is more important is that instead of building houses he created a system for the training of surgeons, and established a train of thought in surgery that will build far more lives than all that would have lived in the houses he might have built.

" But it would not be unfair to say that he began to develop his real stature at Buxton, England. In case it may be wondered what college or training school of surgery exists at Buxton, I may mention that in the 1914–18 war a great hospital centre existed at Buxton. There Edward Gallie, in addition to healing broken limbs and bodies, studied and successfully entered for the Fellowship of the Royal College of Surgeons of England. And it is to the eternal credit of Surgical Royal Colleges that they discriminate neither between universities nor between one nation and another. So Edward Gallie returned to Canada a Fellow of the Royal College of Surgeons of England. And he became Dean of the Medical Faculty of Toronto University, and later he became Professor of Surgery in that same University of Toronto. He applied an alert and observing mind to the correlation of scientific experiment with clinical observations: he did this in the fields of tendon fixation, of bone grafting and re-formation and of the transference of fascia as a living tissue. He built up a great School of Medicine in the city of Toronto, and he developed a system of training young surgeons which became known as the ' Gallie system.' Not unnaturally his hard working, though enthusiastic and devoted, pupils became known as the ' Gallie Slaves.' He raised in Toronto a standard of Surgery which became renowned throughout his own land and throughout the North American Continent, and which is unsurpassed in any other country to-day. In 1947 he again visited the English Royal College—this time to receive its highest distinction, the Gold Medal; and it is a mark of the man that as he stood looking at it in his hand, Edward Gallie said: ' This is one of the events you would like your mother to see.' It is well said that his gifts to the world are innumerable, that his willing ' slaves ' cover the earth, and that his devotion to the science of healing, as well as his contributions to the advancement of our art, deserve the highest honour it is in our power to bestow.

" The Royal College of Surgeons of Edinburgh has a long and honourable history: it has a proud and glorious tradition, and assuredly it will have a great and fruitful future. That which we do here to-day must undoubtedly be witnessed and approved, not only by the distinguished audience here present, but also by the shades of great Fellows of the past, and even by the spirits of those to come. Therefore, Mr President, to you, who so fittingly represent this immortal company, I present with confidence, and with no little pride, William Edward Gallie."

In reply Dr Gallie spoke for all the new Honorary Fellows and he did so in delightfully chosen phrases with a Canadian informality that was just perfect. It was clear that both he and his wife had been deeply affected by his presentation and by the whole proceedings. It was also clear that Dr Gallie had been supremely pleased to be photographed between the Black Douglas and the Lord High Chancellor of Great Britain.

Finally, illuminated addresses of congratulation were presented on behalf of the University of Edinburgh by Sir Edward Appleton, the Royal College of Physicians of Edinburgh by Sir Stanley Davidson, the Royal Faculty of Physicians and Surgeons of Glasgow by Dr A. L. Goodall, the British Orthopaedic Association by Professor Bryan McFarland, and the Executive Committee of the *Journal of Bone and Joint Surgery* by the British Editor. Readers of this Journal ought to know that the address from the Journal was delivered by Sir Reginald Watson-Jones with spirit and with sincerity.

It was fortunate that space allowed for ladies to be present at the Conferring of Honorary Fellowships. Many had been at the Service in St Giles' Cathedral and it must be recorded that their presence added very greatly to the grace and elegance of the proceedings. The good reporter would be able to describe what they were wearing, but on this occasion it must suffice to say that they all looked perfectly charming. It is not in the circumstances invidious to distinguish Mrs Mercer and Mrs Gallie who caught the eye not only because they looked exactly right, but also

because of their obvious anxiety while their respective husbands were speaking, and their relief when they recognised the proper words and phrases duly emerging. The whole affair had in it something of the spirit of Edinburgh Castle as it watches over the city. It was impressive and strong and splendid yet at the same time serene and gracious. And that perhaps is the note on which this account might well terminate, with a final word of congratulation to the College Council and particularly to its secretary, Professor G. I. Scott, who had laboured so assiduously and so successfully.

The Royal College of Surgeons of Edinburgh had rendered tribute to the past; it had given praise to men of great distinction in their service to the Community by conferring on them the highest honour possible; and it had declared its determination to preserve and enhance its heritage in Surgery. It had lived up to its heraldic motto "*Floreat Res Medica.*"

JOURNAL OF THE ROYAL COLLEGE OF SURGEONS OF EDINBURGH

It is with great satisfaction that we have received a copy of the first quarterly number of the first volume of the *Journal of the Royal College of Surgeons of Edinburgh*, published recently on the occasion of the 450th anniversary of the foundation of the College. How right it is that this ancient though increasingly active Corporation should remind its fellows of traditional history and at the same time inform them of recent progress in surgery under the editorship of John Bruce and the chairmanship of the President of the College, Professor Walter Mercer.

This first issue, so excellently produced, includes a coloured portrait of James IV King of Scots who in the fifteenth century granted a Charter, and an appreciation by the Histiographer-Royal in Scotland of the monarch who in the words of His Royal Highness the Duke of Edinburgh: "acquired his own dental forceps and paid his courtiers to suffer the honour, surely a unique one, of having their teeth pulled by their King—and he was skilled at the letting of blood." It includes too an account of the conferment of Honorary Fellowship of the College on the Duke of Edinburgh himself; on the Right Honourable the Viscount Kilmuir, Lord High Chancellor and Keeper of the Great Seal of England; His Grace the Duke of Hamilton, premier peer in the Kingdom of Scotland, whose achievements as a Royal Air Force pilot included that of being the first to fly over the summit of Mount Everest; the Earl of Rosebery who is a descendant of Gilbert Primrose—a president of the College in the sixteenth century; the Right Honourable W. S. Morrison, Speaker of the House of Commons; Sir Edward Appleton, Vice-Chancellor of the University of Edinburgh; the Very Reverend Charles Laing Warr, Chaplain of the College; and Professor William Edward Gallie of Toronto.

There are important surgical contributions of academic and clinical significance, and no more need be said than that if this new Journal of the Royal College of Surgeons of Edinburgh maintains the standard of its first issue there will be redounding credit to an ancient and active College, to Britain, to the Commonwealth of British Nations, and to Surgery. EDITOR.

VISIT OF AMERICAN AND CANADIAN TRAVELLING FELLOWS TO GREAT BRITAIN

The most recent travelling fellows of the American, British and Canadian Orthopaedic Associations sailed from New York on the "Queen Elizabeth" on April 20, 1955, a lovely, sunny day. There were five of us: Albert B. Ferguson, Jun., of Pittsburgh, Charles F. Gregory of Indianapolis, J. Leonard Goldner of Durham, North Carolina, Daniel C. Riordan of New Orleans, and Cameron S. Allen of Vancouver. The Atlantic crossing was a very fine one with clear weather and an exceptionally calm sea. It was unusually pleasant to just rest, talk and eat.

On arriving at Southampton we were met by the president of the British Orthopaedic Association, Professor Bryan McFarland, and by the honorary secretary, Mr J. I. P. James. We later travelled by train to London, where we stayed for the first week.

We initially visited the Royal College of Surgeons and that evening there was a welcoming banquet by the Royal College and the British Orthopaedic Association. It was a superb and stimulating experience to visit the Royal College with its rich historical background, linked with the famous names from the past, as well as the present.

The following day we began the tour in earnest, and the first day was very typical inasmuch as there was a very full and interesting programme presented by the various members of the orthopaedic staff, and the other associated departments of the hospital. Each speaker was well informed on his subject, and usually spoke without notes, with clarity and freshness, and then proved his points by having an excellent follow-up with x-rays, as well as presenting the patient. All the patients were pleasant, very proud of their surgeon, and very glad to have come to the hospital for the re-evaluation. At the end of each paper there was quite a free and frank general discussion.

The Royal College of Surgeons of Edinburgh

from the

British Orthopaedic Association

The British Orthopaedic Association congratulates the Royal College of Surgeons of Edinburgh on its enduring support to Surgery throughout four hundred and fifty years and trusts that this long record and great tradition will be maintained for ever

Bryan McFarland — President
British Orthopaedic Association

Reginald Watkins-Pitchford — Past-President
British Orthopaedic Association

Philip Wiles — President-Elect
British Orthopaedic Association

June 1955

About May 4 we flew to Paris, where we were the guests of the British Orthopaedic Association. This meeting was held in the beautiful new building of the Medical School of the University of Paris. We enjoyed the meeting tremendously and we found Paris to be a fascinating city. Our British confreres very knowingly had allowed us an extra two days in this city, during which we were entirely on our own.

This short respite, however, was soon ended and we returned to London by air and began quite a hectic cross-country travel. We travelled mostly by train, visiting in succession the foremost orthopaedic centres of England and Scotland. The various centres that were visited included Liverpool, Oswestry, Manchester, Edinburgh, Bridge of Earn, Glasgow, Birmingham, Harlow Wood, Sheffield, Oxford, Bath, Bristol and Exeter. In every instance we were accorded a very warm welcome.

American-Canadian Travelling Fellows in Orthopaedic Surgery to Great Britain 1955

Standing—Charles F. Gregory (Indianapolis), Albert B. Ferguson, Jun. (Pittsburgh).
Sitting—Daniel C. Riordan (New Orleans), J. Leonard Goldner (Durham, North Carolina), Cameron S. Allen (Vancouver).

In the late afternoon we visited places with historical backgrounds, such as libraries, museums, castles, cathedrals, or congregated at lovely private homes. In the evening we usually attended a dinner meeting, so that we could then visit with all the surgeons and carry on our interesting discussions. These evenings were most remarkable for the spirit of good comradeship.

It would not be fair in a brief description to single out any specific centre. One of the most impressive points was the place that rehabilitation plays in British orthopaedics. In this respect the rehabilitation centres have been well organised, and this is especially true in the case of the rehabilitation of the permanently disabled individual.

The final event was a most enjoyable banquet and dance at the Savoy Hotel. It included other members of the A.B.C. Club, with their wives, and was under the able presidency of Sir Reginald Watson-Jones.

We enjoyed our travelling fellowship to the utmost. We are all most grateful for this opportunity of visiting Great Britain. It was a very condensed and a very busy time, but we would not have it changed. There was the right mixture of work and play, and the informal gatherings allowed us to have a free interchange of ideas and opinions. A great deal was learned about their sound approach to orthopaedic surgery. We can now more fully understand their wonderful tradition and culture. We have been pleasantly embarrassed by the peerless British hospitality, but most important of all we believe that we have made wonderful friendships. C. S. Allen.

A group of American and Canadian members of the A.B.C. Club photographed with Professor R. I. Harris at a recent meeting in the United States. *Standing*—J. H. Allan, L. R. Straub, Carroll Larson, Verne T. Inman, Hugh Smith, F. P. Patterson, Benjamin E. Obletz, Cameron S. Allen. *Sitting*—W. H. Bickel, S. Benjamin Fowler, R. I. Harris, D. W. Blanche, J. J. Fahey.

UNIVERSITY OF OXFORD

NUFFIELD ORTHOPAEDIC CENTRE

The following lectures have been arranged for the Michaelmas Term 1955.

October 13. *Mr F. G. Allan*—Leg equalisation with special reference to bone lengthening.

October 27. *Professor C. D. O'Malley*—Contributions towards the discovery of the circulation of the blood in the sixteenth century.

November 10. *Professor J. Trueta*—Considerations on the present treatment of skeletal tuberculosis.

November 24. *Dr L. Guttman*—The initial treatment of traumatic paraplegia.

SCIENTIFIC SOCIETIES AND REGIONAL ORTHOPAEDIC CLUBS

BONE AND TOOTH SOCIETY

The Society held a "Casual Communications" meeting at the Institute of Orthopaedics, London, on June 9, 1955. Dr H. R. Perkins was in the chair.

Variations in severity in the nail-patella syndrome—*Dr J. H. Renwick* said that the nail-patella syndrome, due to a dominant gene, included a hypoplasia or absence of the patellae, dystrophic nails, posterior dislocations of the heads of the radii, iliac horns and lordosis. It had been found that the severity of the affection in the offspring was not correlated with that in the affected parent, whereas between affected sibs a correlation of about 0·5 was found in the degree of severity. It was therefore tentatively postulated that the severity was largely genetically determined by the particular modifying allele which happened to be present at the nail-patella locus itself; in other words that it was determined by the partner of the nail-patella gene.

Techniques for the histological identification of osteoid tissue—*Dr P. Meyer* had studied the effects of different methods of decalcification and staining on the histological identification of osteoid tissue in material from rachitic dogs, rats, and human subjects. Decalcification included formic-citrate mixture, nitric acid, Muller's fluid and E.D.T.A. Celloidin and paraffin sections were stained with haematoxylin and eosin, with connective tissue stains and by the von Kossa technique. Osteoid tissue was most easily identified in decalcified material after the use of Muller's fluid and E.D.T.A. There was complete correlation between the appearance of osteoid tissue in stained sections of bone decalcified by all four methods, and the findings in ground sections of perspex-embedded tissues, which were also compared with corresponding microradiographs.

Correlation between growth rate and changes in the epiphysial plate—*Dr A. Fahmy* discussed the correlation between growth rate (serial radiography) and objective quantitative histological changes in the upper tibial epiphysial cartilage place (E.C.P.) of albino rats. The total thickness of the E.C.P. and of the so-called "proliferative" and "hypertrophic" layers were measured, the latter being estimated by arbitrarily dividing the plate at the level where vacuolation just appears in cartilage cells. Extremely significant (P less than 0·001) correlations existed between growth rates and E.C.P. and layer thicknesses under optimal growth conditions, or when all osteogenic processes were gradually and equally affected (e.g., sub-optimal diet, methionine deficiency, cortisone administration) but not in rachitic and irradiated plates. The correlation was useful in assessing how factors affecting endochondral osteogenesis worked.

Composition of medullary bone of hens on a low-calcium diet—*Mr T. G. Taylor* and *Dr J. H. Moore* had found that successive eggs laid by hens on a low-calcium diet contained diminishing shell calcium. After six eggs the loss of skeletal calcium rose to 38 per cent. Meanwhile phosphorus lost from the bones was not all excreted, and might have passed to the soft tissues. Medullary bone, purified from non-osseous matter by centrifugation in carbon tetrachloride, showed little change in calcium and phosphorus content, increases in magnesium, sodium and potassium and a decrease in citrate as more eggs were laid on the deficient diet.

Leontiasis ossea—*Mr E. D. D. Davis* described the morbid anatomy of leontiasis, including thickened periosteum with increased vascularity, and the simultaneous formation and reabsorption of new bone. Compensatory ossification to give rigidity to abnormal soft bone was usual.

SOUTH-EAST METROPOLITAN REGION ORTHOPAEDIC CLUB

The Club visited Southlands Hospital, Shoreham-by-Sea, on June 25 at the invitation of Mr J. Addison, who produced a large and varied collection of clinical problems for discussion.

Osteoarthritis of the hip—Discussion centred mainly on the treatment of bilateral osteoarthritis of the hip, though considerable variety was manifested in the choice of method for arthrodesis in unilateral cases. *Mr J. C. Adams* favoured the Norwich V arthrodesis. *Mr G. C. Lloyd-Roberts* reported excellent results by the method reported from Pyrford, and *Mr T. T. Stamm* reported 93 per cent of bony fusion using a modified Watson-Jones method with post-operative splintage in a short plaster spica. This method was also preferred by *Mr J. Addison*. Several cases of arthrodesis by central dislocation were shown. In one of these a pseudarthrosis was present with a small range of painless flexion, and the possibility of using some form of central dislocation for arthroplasty was considered.

In discussing bilateral osteoarthritis *Mr E. A. Lindsey* supported an operation on the lines of the Whitman reconstruction and made a plea for the use of metal rather than plastic materials if any type of prosthesis had to be used. He preferred an arthroplasty created by living bone without the interposition of any inert material. *Mr H. L.-C. Wood* thought that bilateral McMurray osteotomy ought to be more widely practised and reminded the meeting of the excellent results of this operation demonstrated at the British Orthopaedic Association meeting at Biddulph Grange two years ago. *Mr J. H. Mayer* put in a plea for the Smith-Petersen operation but *Mr J. C. Adams* replied that in his view all forms of cup arthroplasty were bad. He attributed most of the failures to deficient blood supply and he now favoured some form of pseudarthrosis by Girdlestone's or Batchelor's operation if arthroplasty had to be advised. *Mr J. S. Batchelor* thought that the best treatment for severe bilateral osteoarthritis was arthrodesis on the worst side and some form of pseudarthrosis on the other. *Mr J. Buck* favoured a splined McMurray-type osteotomy using a nail-plate with immediate mobilisation. *Mr T. T. Stamm* suggested that we must continue to investigate the cause of pain in osteoarthritis and after arthroplasty operations. In his view pain might be due to soft tissues being compressed or stretched, or to shearing strains. He believed that many methods could be used according to the individual case and he emphasised the need for careful technique to achieve the best results. He himself was experimenting with a new type of prosthesis in the hope of achieving an arthroplasty providing a large weight-bearing area without shearing strains.

Other cases—*Mr J. Addison* demonstrated a method of arthrodesis of the shoulder joint using Charnley's compression clamp. Sound bony fusion had been achieved without plaster fixation, though he had found it necessary to fix the shoulder in less abduction than is usually advised.

Mr Addison showed two cases of knee arthroplasty using a special bi-convex perspex prosthesis. One of these had been done about four years, and the patient had retained an excellent range of movement. The other presented a more limited range but the patient was well pleased with the result.

A boy of sixteen was demonstrated with slipped upper femoral epiphysis, slipped lower tibial

epiphysis and vertebral wedging with kyphosis. For treatment of the slipped upper femoral epiphysis in recent cases *Mr Batchelor* emphasised that epiphysial osteotomy could give good results, but not in old ones with much new bone formation. He emphasised that enough bone must be removed from the femoral neck to allow easy reduction without tension.

Some discussion was provoked by a case of bilateral stress fractures of the femoral necks. In this particular case the serum calcium was moderately raised but serum organic phosphates were normal. Many present felt that more than one variety of stress fracture might occur in the femoral neck and *Mr W. Laurence* suggested that some of these cases might be associated with a congenital type of coxa vara.

A child with unilateral hip dislocation following infantile suppurative arthritis raised the question of how these hips dislocate. *Mr Batchelor* suggested that there might be a pathological fracture of the femoral neck or dislocation of the epiphysis from the neck.

A series of ruptured extensor pollicis longus tendons was shown after repair. *Mr J. Buck* favoured the nylon bridge operation described by Trevor, and *Mr Adams* favoured the use of an extensor indicis transplant. Extensor carpi radialis transplants were unsatisfactory because the tendon excursion was not great enough. *Mr Austin Brown* had found that the use of extensor indicis tendon resulted in some weakness of extension in the index finger.

NORTH-EAST METROPOLITAN REGIONAL ORTHOPAEDIC CLUB

A meeting of the North-East Metropolitan Regional Orthopaedic Club was held at Black Notley Hospital on Saturday, July 16, 1955.

Clinical cases—*Mr H. Osmond-Clarke* showed a case of vertical talus in an infant successfully treated by open reduction of the talo-navicular dislocation and transplant of the peroneus brevis tendon into the neck of the talus.

He also carried out a demonstration operation of by-pass graft for pseudarthrosis of the tibia.

Mr D. M. Dunn showed cases of coeliac rickets, polyostotic fibrous dysplasia and intrauterine dwarf with congenital dislocation of the hip.

Mr M. C. Wilkinson showed cases of skeletal tuberculosis, illustrating the effectiveness of early partial synovectomy, curettage and free drainage of tuberculous foci accompanied by chemotherapy.

Paper—*Dr W. S. Tegner* discussed modern ideas in the treatment of rheumatoid arthritis, and outlined the proposed pilot scheme for sanatorial treatment of such cases.

EAST ANGLIAN ORTHOPAEDIC CLUB

A meeting of the East Anglian Orthopaedic Club was held on March 26, 1955, at the West Suffolk General Hospital with Mr G. K. McKee in the chair.

Fracture of the cervical spine—*Mr D. J. Martin* showed a series of cases of fracture of the cervical spine including one fatal case in which the articulated specimen was available with sections of the cord. There were also two cases treated by wiring of the spinous processes.

Children with limps—*Mr B. J. Batt* showed a series of cases of children presenting with limps which on investigation proved to be due to aseptic necrosis of the femoral head following a partial fracture of the neck nine months before, a simple cyst of femur treated by opening and curetting, and sclerosing osteitis of the shaft of the femur of unknown cause.

Osteochondritis—*Mr E. C. Scanlon* demonstrated a number of cases of osteochondritis of various bones in children, and discussed the treatment and prognosis especially in connection with Perthes' disease, making a plea for continued bed rest and prolonged relief from weight bearing.

Acute epiphysitis—*Mr A. B. King* showed films of a child who had been admitted with acute pain in the hip and a raised temperature. In the course of two weeks an area of rarefaction had appeared in the neck and increased density of the head. This was thought to be an example of acute epiphysitis.

Hallux valgus in adolescence—*Mr Martin* discussed the treatment of hallux valgus in adolescence and showed two cases of double osteotomy of the first metatarsal. In an interesting discussion it was generally agreed that operation should be avoided altogether if possible and there was no agreement on the best type of operation.

Ossifying haematoma of the quadriceps—*Mr D. C. Tennant* showed the case of an ossifying haematoma of the quadriceps in a man who attended for treatment nine weeks after injury. He discussed the mechanism of its production. In discussion which followed prevention was considered, the main suggestion being real rest after the injury, which should be taken seriously.

A further meeting was held on June 4, 1955, at the Royal Air Force Hospital, Ely, with Mr G. K. McKee in the chair. Clinical cases were demonstrated.

Osteomyelitis of clavicle—*Squadron Leader K. R. J. de Belder* described a case of a young man who had an acute staphylococcal sore throat which was followed by a diffuse swelling in the neck about the clavicle. The acute symptoms settled, but the swelling over the clavicle persisted and was sometimes associated with aching and later with a spasmodic torticollis. Radiographs showed a sequestrum in the middle of the clavicle. This was removed through a mass of inflammatory tissue. In discussion it was suggested that the infection had arisen from direct spread from infected cervical glands rather than from blood-borne infection.

Multiple injuries from jet intake accidents—*Wing Commander D. M. Keir* described three cases of men who had been sucked into the air intake of jet engines. There was a certain similar pattern in all cases. The men sustained extensive head injuries, fractured ribs and haemo-pneumothorax and fractured spines. According to the man's position as he went in there were sometimes fractures in the limbs as well. These patients presented great difficulties in treatment because they were *in extremis* and there was inevitable delay before the injuries to limbs could be tackled.

Wing Commander Keir described the result of ejector seat accidents and showed one man who had been propelled 30 to 40 feet into the air, landing on a concrete runway. He sustained a compound fracture of the calcaneum, fractured neck of femur, a supracondylar fracture of the humerus, and a fractured base of skull with cerebral irritation.

Recurrent dislocation of elbow—*Squadron Leader J. F. Stone* described a case of recurrent dislocation of the elbow associated with a former fracture of the coronoid process. Treatment was by insertion of a piece of bone into the tip of the coronoid.

Radiographs of orthopaedic interest—*Wing Commander M. H. Phillips* showed a series of radiographs of orthopaedic interest and there was a discussion on the diagnosis of a number of anomalous findings.

Fracture of scaphoid—*Squadron Leader Griffiths* discussed the treatment of fracture of the scaphoid and showed the result, in a long-standing case with considerable degeneration, of removal of the styloid process of the radius.

AUSTRALIA

ROYAL AUSTRALASIAN COLLEGE OF SURGEONS

The diary of the College includes a number of interesting events, prominent among which is the visit of Professor R. I. Harris, of Toronto, who is the Sims Commonwealth Travelling Professor for 1955. The programme shows that Professor Harris had a very full itinerary in Australia and New Zealand in July, August and September.

At the General Scientific Meeting, held in Sydney on August 16–19, the first subject for discussion was injuries of the hand, by Mr D. Officer Brown, Mr A. R. Wakefield and Mr John Lahz. Professor R. I. Harris contributed papers in the symposia on the treatment of fractures of the long bones by intramedullary fixation, and structural changes in the lumbo-sacral spine. Visitors from Britain included Professor B. W. Windeyer, of London, who spoke on chordoma, and Sir Stanford Cade, who read papers on several aspects of cancer.

NEW ZEALAND

BIRTHDAY HONOURS 1955

We are glad to record that Mr James L. A. Will, of Christchurch General Hospital, has received the C.B.E. (Civil Division).

EAST AFRICA

ASSOCIATION OF SURGEONS OF EAST AFRICA

In a recent report Mr W. H. Kirkaldy-Willis, the honorary secretary, outlines suggested methods of improving higher surgical training in East Africa.

The Association is planning to hold regional meetings three or four times a year in addition to the Annual General Meeting. The next annual meeting will take place in Nairobi in February 1956.

DENMARK

DANISH ORTHOPAEDIC ASSOCIATION

A meeting was held in Copenhagen on June 11–12, 1955.

Injuries to the brachial plexus in adults—*Dr E. Hj. Larsen* (Aarhus) reported fifty-six cases from the orthopaedic hospital, Aarhus. Forty-five were in males and eleven in females. Motor-cycle

accidents were the most frequent cause. Thirty-one cases were of the "upper" or "Duchenne-Erb" type. One-third of these presented an almost complete recovery within a year of the accident. No improvement was shown in 10 per cent. Twelve cases gave evidence of injury to the whole plexus. Forty-two per cent of these showed no recovery; the rest showed improvement in regions innervated by the upper roots. Thirteen cases were atypical; two-thirds of these (8 per cent) presented improvement or complete recovery and about one-third no alteration. In twelve cases an operative exploration of the plexus was done without any improvement.

The use of homologous transplants from the bone bank—*Dr E. Thomasen* (Aarhus) reported 228 operations performed since 1949 at the orthopaedic hospital, Aarhus, with the use of human bone from a deep-freeze bone bank. Bone was obtained mainly from amputations at other hospitals, and was fetched directly from the operation theatres and immediately frozen by carbonic acid before transport to the bank. Most of the operations were fusions of the spine for scoliosis or other spine lesions. Ninety per cent healed without any complication and a further 6 per cent showed only slight unwanted reaction. *Dr Thomasen* was satisfied with the osteogenetic property of the transplant.

Congenital pseudarthrosis of tibia and bowing of diaphysial bones—*Dr E. Thyge Madsen* (Copenhagen) gave a survey of the literature concerning etiology and treatment of pseudarthrosis and congenital angulation of the tibia and other diaphysial bones. He believed Duraiswami's experimental studies of skeletal abnormalities suggested that the conditions were of identical nature. Concerning treatment of pseudarthrosis of the tibia, he believed McFarland's "by-pass" bone grafting to be the best. Radiographs of cases were shown.

Treatment of spastic paralysis—*Dr P. G. K. Bentzon* (Aarhus) gave his ideas on the role of training and physiotherapy of spastic children versus orthopaedic treatment. He believed that the latter was most helpful, and that it should not be dismissed entirely as advocated from some clinics.

HOLLAND

ACTIVITIES AND DEVELOPMENT IN THE FIELD OF ORTHOPAEDICS

Our Corresponding Editor, Dr J. D. Mulder, writes: In September 1952 Dr E. H. la Chapelle left Amsterdam for Utrecht to become university lecturer in orthopaedics and chief of the orthopaedic section in the surgical department of the University Hospital (Stads- en Academisch Ziekenhuis). The director of the surgical department is the professor of general surgery, Professor J. F. Nuboer. At the same time he accepted an appointment as orthopaedic surgeon to the rehabilitation centre "De Hoogstraat" at Leersum, which opened its doors in 1953.

Dr la Chapelle was succeeded as head of the orthopaedic department of the Wilhelmina Gasthuis in Amsterdam by Dr O. Verbeek, who has at the same time accepted a position as chief of the orthopaedic section of the university surgical department under Professor I. Boerema. Amsterdam however does not have an official lecturer in orthopaedics although Dr O. Verbeek gives two lectures a month to the Amsterdam students of medicine, whereas Dr la Chapelle gives two lectures a week to students in Utrecht.

At the University of Groningen Dr W. Mol became an official lecturer in orthopaedics in January 1954. He gives two lectures a week. Dr Mol also has the supervision of thirty "orthopaedic beds" in the university surgical department under the direction of the professor of general surgery, Professor L. D. Eerland.

The University of Leiden is the only one in this country which does not have an orthopaedic department, the Anna-Kliniek being a private hospital not related to the university. Although the independent position of the Anna-Kliniek is a most valuable thing and far to be preferred to the position of orthopaedics in other towns mentioned above, it is strange that there should be no orthopaedic department at a famous University like Leiden. Dr C. P. van Nes still holds his appointment as a "privaat docent" (private teacher at the university) in orthopaedics at Leiden University and gives clinical lectures for one hour a week.

In the scientific field for the last three years, mention should be made of the work of Dr W. Mol on stapling for scoliosis. He started using staples for this purpose in 1952 and gave a preliminary report on five cases in the February session of the Netherlands Orthopaedic Society in 1953.

In the session of November 1954 of this Society Dr W. C. Meiss of The Hague reported on the use of deep-frozen calf's fascia in reconstructive operations. He had used this material twice in knee arthroplasty. One case concerned a twenty-years-old patient with nearly complete ankylosis from rheumatoid arthritis, in whom the result was a range of mobility from complete extension to 90 degrees flexion. Deep-frozen fascia was also used twice for the repair of a torn calcaneal tendon and once in a tear of the patellar ligament.

In March 1955 Dr L. N. M. Coene reported in the Netherlands Orthopaedic Society on results of epiphysiodesis around the knee joint. Twenty-five patients had been operated upon in the orthopaedic department of the Wilhelmina Gasthuis in Amsterdam, twenty-two on account of a short leg and three for a valgus or varus deformity. Phemister's method was used in sixteen patients at an average age of fourteen years. In the others, Blount's staples were used. The results of calculations according to White and Green-Anderson did not agree amongst each other, nor with the eventual outcome of the operation. The best age was considered to lie between ten and twelve years. Epiphysiodesis of both femur and tibia at the knee often led to over-correction. In the discussion Dr van Nes said that he usually operated on the tibial side only. He used the Phemister method at the age of ten to twelve years. Operation on the femur sometimes caused irritation of the knee joint, which was not caused by metallosis but by haemarthrosis (see the report by Himpe, L., and Spering, H., on 265 epiphysiodeses carried out under Dr van Nes (1954), in Acta Orthopaedica Belgica, **20,** 528).

In June of this year a session of the Netherlands Orthopaedic Society was held in the Anna-Kliniek, Leiden. Dr Th. Boesman reported on myelography with the aid of a water soluble contrast medium. His results will be published later. Dr S. Bouwer spoke on results of the use of calf bone in spinal fusion and other bone grafting operations. He was most disappointed about the qualities of calf bone as it did not promote bone formation and did not unite with the host bone. Altogether forty-three cases were reviewed in which calf bone had been used, thirty-seven of which were cases of spinal fusion.

Dr F. Duijfjes reviewed the results of transplantation of autogenous cancellous bone from the ilium in post-traumatic osteomyelitis with draining sinuses (ten cases). Cancellous chips were accepted even in the presence of infection and united promptly with the host bone. Granulation tissue appeared to invade the chips, sequestration was a rare exception and even then only a few chips were extruded, as most of the implanted material remained in place and eventually fused with the surrounding bone.

The writer had treated seven patients with scaphoid pseudarthrosis after the method of Matti-Russe. A graft of cancellous iliac bone is implanted through a small volar incision. Immobilisation is continued for four months. Bony union with good function was obtained in six cases. The method seems definitely superior to previous ways of treatment. Another communication by the writer was on comparative results of the Eicher prosthesis in surgery of the hip joint and resection of the femoral head and neck after Girdlestone. His impression was that the intramedullary prosthesis, which had been used since 1952, gave the best results in post-traumatic cases (aseptic necrosis of femoral head, fracture of the neck with resorption of the neck). Its use in osteoarthritis is of limited value. Resection according to Girdlestone is at present the best answer in failure of a hip arthroplasty. Pain usually disappeared nearly completely, the patient was able to walk over small distances with a limp—and a stick—and a wide range of painless mobility was always obtained. The use of the Judet prosthesis was discarded completely on account of the disappointing long-term results.

BELGIUM

TENTH BELGIAN CONGRESS OF SURGERY

The tenth Belgian congress of surgery, organised jointly by the Belgian Surgical Association, the Belgian Surgical Society and the Belgian Orthopaedic Society, will be held at Knokke on May 10–12, 1956. The main topic for discussion will be post-traumatic osteoporosis. Those wishing to read papers or to take part in the discussion should communicate with the Secretary of the Congress, Dr M. Bélenger, 99 Rue Jourdan, Brussels.

INTERNATIONAL SOCIETIES

WORLD CONFEDERATION FOR PHYSICAL THERAPY

The Second Congress of the World Confederation for Physical Therapy will be held at the Hotel Statler, New York City, on June 17–23, 1956. The programme has been developed around the theme "Health, a Strong Force for World Understanding—The Role of the Physical Therapist," and the Congress will include scientific papers delivered by leading physical therapists and physicians from America and other countries, reports by official observers from other nations, scientific and technical exhibits, and study visits to several local departments of physical therapy.

Book Reviews

KINESIOLOGY OF THE HUMAN BODY under normal and pathological conditions. By Arthur STEINDLER, M.D., Hon.F.R.C.S. (Eng.), F.A.C.S., Professor of Orthopaedic Surgery (Emeritus), State University of Iowa, Head of Orthopaedic Department, Mercy Hospital, Iowa City, Iowa. $10\frac{1}{2} \times 7$ in. Pp. xxiv + 708, with numerous figures. Index. 1955. Springfield, Illinois: Charles C. Thomas, Publisher. Oxford: Blackwell Scientific Publications. Price £7, 2s. 6d.

This most recent work of Arthur Steindler is something of a landmark in orthopaedic surgery in that it may well establish biomechanics as a complete subject in its own right, necessary for basic training, and perhaps worthy even of a professorial chair. Were he never to have done more than this, Steindler could be justifiably proud of his life's work. Doubtless there will be arguments on the best name for this new province of the human mind, and " kinesiology " to many may seem not as good as " biomechanics," because kinetics to the physicist and engineer is only one part of mechanics (i.e., statics, kinetics and hydraulics). Some of the allure of orthopaedic surgery is the opportunity it offers the surgeon to make basic observations relevant to normal physiology; observations that are denied to the physiologist and the anatomist working on cat or corpse.

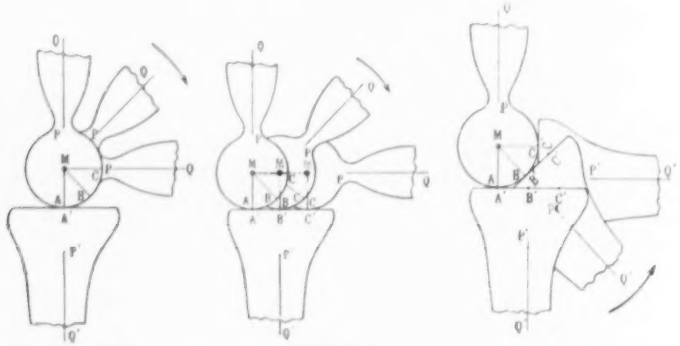

FIG. 5 FIG. 6 FIG. 7

Figure 5—Joint motion—gliding of first order (Fischer). Figure 6—Joint motion—gliding of second order (Fischer). Figure 7—Rocking motion—contacting surfaces change position of both joints consecutively (Fischer). (Reduced by 20%.)

In every page Steindler imparts the thrill of the investigator detecting in some biological phenomenon the hint of a law that might show some unity with Newtonian theory of the universe. In these pages one realises that orthopaedic surgery stands in relation to general surgery much as physics does to chemistry.

To enjoy this book it is best dipped into from time to time " to see what Steindler says about so and so," and the dip will be fruitful either directly in the text or through the references which the writer's encyclopaedic knowledge makes available. No orthopaedic surgeon need be ashamed to confess that he has been unable to read it through from cover to cover as a bedtime task (even though the dust-cover recommends it as reading matter for dentists, chiropodists and osteopaths). In arrangement, the book starts with the statics of the skeleton, in particular the theory of beams and columns, and passes to the strength of materials as exemplified by the physical properties of bone, muscle, tendon, cartilage and fascia. In kinetics the structure and function of joints is outlined, leading to a discussion of the equilibrium of the body in the light of these principles, starting with the trunk and working through the limbs to end in a chapter devoted to the gait in health and in disease, for the study of which Steindler has long been famous.

Throughout these chapters there are numerous clinical digressions, particularly in relation to the surgery of infantile paralysis, most of which are valuable and interesting. Yet one cannot help feeling that much of the interposed clinical work is rather out of place in a text-book the title of which relates essentially to an abstract subject, and that Steindler has already handled these matters better in the context of his postgraduate lectures. In this one suspects the pressure of the publisher and that, while Steindler himself would like to be loose from earth-bound ties and be free to elaborate a mathematical theme, he has been made to remember the chiropodists, and even try to teach them anatomy as well as orthopaedics. Surgeons who are good anatomists will thus discover irritating anatomical banalities, such as hackneyed illustrations of the bones of the foot (with tarsal bones all carefully named!) when at the same time excessive brevity in the kinesiological paragraphs tend to leave the reader wondering whether enough explanation has been offered or whether he himself is at fault in not being able to understand it more easily. Thus, not many readers for instance will be able to understand the most interesting illustrations taken from Pauwels (page 17) if they do not already happen to know how engineers demonstrate stresses by optical techniques. There are times when one feels that the author is revelling in complexity when the problem is screaming for simplification. An instance of this is seen in the reproduced illustrations where Figures 5, 6 and 7 are given to illustrate "gliding of the first order," "gliding of the second order" and "rocking," without any more explanatory help in the text. If we ponder we see that Figures 6 and 7 are fundamentally the same, the diagrams being different only in that the femur is flexed on the tibia in one and the tibia flexed on the femur in the other: so why perpetuate the bogus phrase "gliding of the second order" if there are only two basic types of motion and these are no more pompous than "gliding" and "rocking"? After puzzling on this matter many readers will fall back exhausted, saying, "anyhow, does it really matter?"

But despite criticisms of this type, which come only from the purist and splitter of hairs, this is a truly helpful work and one which passes the true test of greatness in that it will stimulate disciples to the production of further work, and no library therefore must be without it.—John Charnley.

AN ATLAS OF MUSCULOSKELETAL EXPOSURES. By H. F. Moseley, F.R.C.S. (England and Canada), F.A.C.S., Hunterian Professor, Royal College of Surgeons of England, Assistant Professor of Surgery, McGill University, Associate Surgeon, Royal Victoria Hospital, Montreal. 11 × 8½ in. Pp. xii + 235, with 75 colour plates and 63 figures. 1955. Philadelphia and Montreal: J. B. Lippincott Company. London: Pitman Medical Publishing Co. Ltd. Price £9.

"This Atlas is designed to present in visual form, with an adequate descriptive text, the detailed anatomic relations of the bones and joints of the face, upper extremity, vertebral column and lower extremity." The visual form chosen is a series of simplified two-dimensional diagrams. In seventy-one plates, each containing from two to ten of these diagrams, the author starts by showing the anatomy of the areas under consideration, building this up layer by layer from the skeleton. Then, by reversing the process, the operative exposure is illustrated, beginning with the skin incision and working by stages down to the bones and joints. The plates themselves are the work of two artists of rather contrasting styles, but both of them use colour diagrammatically as a means of presenting anatomical facts and make no attempt to reproduce accurately the actual colours of the tissues.

No such book can be wholly satisfactory. None of the anatomical atlases and none of the atlases of operative orthopaedic surgery that have been produced in the past have found much favour with teachers of anatomy or of orthopaedic surgery nor, one suspects, with the majority of surgeons who use them to learn rather than to teach. The presentation of these facts and techniques in two-dimensional diagrams somehow fails to "come off." This is a pity, for such atlases involve a vast amount of work in their preparation and, if many colour plates are included, they become very costly. This book, the latest attempt at such presentation, succeeds no better than its predecessors, though its producers have spared neither labour not cost. Indeed, the cost could surely have been reduced in this instance. There are so many diagrams on some of the coloured plates that their very profusion destroys clarity. Is so extensive an illustration as plate 5 really necessary to show the anatomy of the sterno-clavicular joint? What is the point of a picture (plate 57) of a fractured femur treated with a plate so slender that its proper place is a museum? What useful purpose is served by plate 61*f*, or 47*c*, or by 42*c*, to mention a few only? The purpose of the frontispiece is quite obscure to me, but that may be my fault.

A good deal of the text could have been omitted without loss. The many pages that are devoted to illustrations of the author's methods of towelling limbs have no bearing on the main purpose of the book. It is claimed that they may " assist the surgical intern staff." But how can they? Do not the interns have to put on towels and stockinette according to the orders of their individual chiefs, and not as laid down in any other surgeon's text-book? Anyone who has the courage to write a book on operative surgery is, of course, an easy target for minor criticisms, but do we really need to be told to avoid nicking the patient's skin during the pre-operative shaving (page 4), or to remove pediculi from the scalp before operation on the face (page 19)? What is an electronic blanket (page 126)? Do we look to books presumably directed to qualified surgeons to learn that " operations on the tibia and the fibula are also required for infective and neoplastic processes at various sites " (page 202), and is there really any evidence that " the knee joint is being explored with ever-increasing frequency " (page 191)? Instances of this sort of thing abound in the text. While the author makes no claim to originality one is a little shocked to see diagrams of incisions in the hand attributed to his own text-book when they are in fact (Fig. 31) almost identical with the well known " right " and " wrong " diagrams of Bunnell.

The book is well produced and the index is adequate, but I fear that it is not a work I can recommend to anyone who wants good value for a very high price.—D. Ll. Griffiths.

CHIRURGIE DE LA MAIN. By Marc Iselin, Chirurgien de la Maison de Nanterre, Chirurgien consultant de l'Hôpital Américain, Membre de l'Académie de Chirurgie. 10 × 7 in. Pp. iii + 626, with 325 figures. Index. 1955. Paris: Masson et Cie. Price: Broché, 4,000 fr.; Cartonne toiles, 4,800 fr.

It is a pleasure to review this book, written by one of the world's most experienced hand surgeons. Marc Iselin has produced the most important work upon this subject since Bunnell's classic volume. It reflects not only his own mature judgment, but is generous in its references to the work of other surgeons. No one engaged in surgery of the hand should neglect to study this

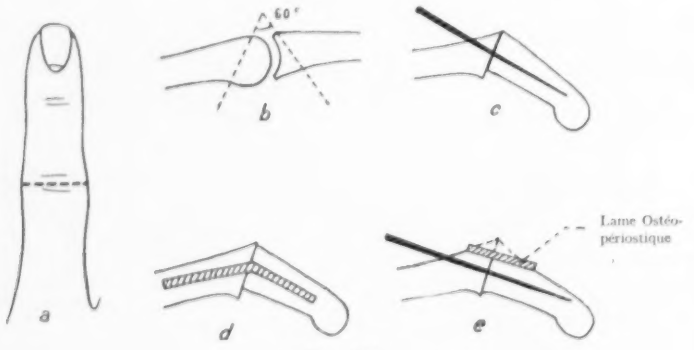

Fig. 104

La résection ankylosante et sa fixation.

a) L'incision.
b) L'angle de coupe = 60°.
c) Fixation par broche (1 ou 2).
d) Fixation par greffon coudé enchevillé dans la médullaire.
e) Fixation par clou plus lame ostéo-périostique (Howard).
Aucun de ces procédés ne donne l'ankylose avec certitude.

book, for there is much for all to learn, and some of the methods described will be new to most readers. The book is well balanced and there are many good illustrations, which is a most important feature in a work which necessarily concerns itself with precise technique. An excellent review has recently appeared in the American volume and a description given of the contents. Comments now are restricted to special points of interest.

The chapter on skin injuries (eighty-six pages) is particularly well done and of real practical value. British surgeons would probably prefer a cross arm flap to the somewhat heroic flaps illustrated in Figures 36 and 63. Corrective operations for bone deformities and joint stiffness are

discussed, including the importance of intramedullary bone grafts for metacarpal fractures. The chapter on tendon injuries (eighty-two pages) is comprehensive and quotes the work of Verdan, among others, which deserves to be more widely known. Iselin gives his own conclusions on the results that may be expected from different injuries and technique in an attractive and thoughtful manner which the reader will appreciate.

Several methods of thumb reconstruction are illustrated, including the Hilgenfeldt technique, the text of which is difficult to obtain. It is a pity that the name of Gillies is not associated with the "cocked hat" procedure, and we should also like to have seen more reference to the excellent work of Littler on pollicisation. The chapter on the sequelae of amputations, and the various ways of replacing the amputated hand, will be read with much interest.

Here then is a book that will take its place among the classics, and we echo the wish of Kaplan when he asks that an English translation be prepared.—R. Guy PULVERTAFT.

FRACTURES AND JOINT INJURIES. By Sir Reginald WATSON-JONES, B.Sc., M.Ch.Orth., F.R.C.S., F.R.A.C.S. (Hon.), F.A.C.S. (Hon.), F.R.C.S.E. (Hon.), Extra-Orthopaedic Surgeon to Her Majesty the Queen, Orthopaedic Surgeon to His Late Majesty King George VI, Director, Orthopaedic and Accident Service, London Hospital, Senior Surgeon, Robert Jones and Agnes Hunt Orthopaedic Hospital, Shropshire, Civilian Consultant in Orthopaedic Surgery, Royal Air Force, British Editor, *Journal of Bone and Joint Surgery*. Volume II. Fourth edition. 10 × 7½ in. Pp. viii + 629, with 904 figures, many in colour. Index. 1955. Edinburgh and London: E. & S. Livingstone Ltd. Price per set of two volumes £6.

I wonder if he who reads this book remembers the words of the Marquis of Reading at our London meeting—Here is a language, rich, beautiful, supple, resonant and strong. But perhaps, too, he who reads will think of the words of Zimmerman—"I have never been in too great a hurry." At last, however, the second volume has arrived, and it is worth waiting for. It is a great book, full of wisdom, excellent in advice, and delightful to read.

On the first page we come across the dictum of the author on the treatment of all injuries—"Many are disabled by recurrent dislocation of the shoulder through neglect to immobilise the first dislocation—a neglect that is foolish in healthy young men." Sir Robert Jones said that no harm came from the immobilisation of the healthy joint; and how true it is, and how good to have this emphasised by this master surgeon of trauma. It is brought home again in the treatment by passive manipulation after a dislocation of the elbow of a violinist who was never able to play a violin again because of a stiff elbow.

Throughout the book basic principles of treatment are laid down to the exclusion of abstruse methods of treatment. Nothing could be better than the simple but tremendously important principles of the treatment of finger injuries. To know the first two pages of Chapter XXIII is to know the treatment of finger and hand injuries. A new operation for the rare recurrent forward dislocation of the ankle is described by using the peroneus longus tendon. Various rather useless methods of dealing with an acromio-clavicular dislocation are detailed, none of which is used by the author and so one would be content with his advice. So, too, in the management of recurrent dislocation of the shoulder, must he be polite and describe so many operations. What does *he* do?

A most excellent treatise on the treatment of fracture of the shaft of the femur is delightfully written and of immense value. This, of course, is of the Liverpool school and it is a pity that it is not more commonly the practice. There would be fewer catastrophes: to choose only one complication, there are too many stiff knees. Excellent advice is given on the prevention of this and it is pointed out that there are three different causes—adhesion formation, fixation of the patella, and fixation of the quadriceps—and all preventable. The Jones method of correcting the displacement in a supracondylar femoral fracture is quite unique, quite effective, and probably the only method that is so.

The summary of the treatment of fractures of the neck of the femur in four age groups should be read by every young surgeon as it is so full of commonsense, but I should have expected, especially from the Liverpool school, that a place might have been reserved for the McMurray osteotomy. One of the best chapters is that on injuries of the trunk, especially the treatment of fractures of the spine with paraplegia, and it is interesting that the paralysed bladder is quite within the compass of this brilliant man.

The book itself is from the House of Livingstone and, having said that, anyone can understand that the production—illustrations, printing and paper—are the best in the country!

A wonderful book and one that the author may well be very proud of, as, indeed, are all his friends.—Walter MERCER.

THE CASUALTY DEPARTMENT. By T. G. Lowden, F.R.C.S., Consultant Surgeon Sunderland Royal Infirmary. 9½ × 6¼ in. Pp. viii + 278, with 134 figures. Index. 1955. Edinburgh and London: E. & S. Livingstone Ltd. Price 37s. 6d.

This book is intended to be a guide to the casualty officer in the medium-sized non-teaching hospital, and to provide him with methods which he can safely adopt in the interest of the patients and an idea of the results he can expect to achieve.

To understand the method of selection of subjects and the relative emphasis it is necessary to quote the writer's definition of the casualty department. "The Casualty Department is a haphazard growth. The extent of its functions varies from hospital to hospital according to the stage of evolution of the other departments for, generally speaking, the Casualty Department deals with everything that nobody else wants." The emphasis is on minor wounds and infections of various kinds. Diagnosis and treatment of closed soft tissue injuries occupy ten of the 273 pages; injury to the knee is dealt with in four sentences; Sudeck's osteoporosis has two pages. Treatment of fractures is not included. Infections of the hand are completely and effectively dealt with and the reader is given satisfactory alternatives of treatment and told what results he should obtain. Thirty pages are devoted to open soft tissue injuries to the hand, including those with skin loss. The fifteen pages devoted to burns and scalds deal principally with the proportion of these injuries that can be treated as out-patients. The chapter dealing with the "cold" case includes a variety from malignant skin lesions to ingrowing toenails. More than a third of the book is devoted to anaesthetics, organisation, disposal and legal problems, and it is an interesting comment on our times that the space devoted to legal considerations equals that for closed soft tissue injuries and burns and scalds put together.

As the author says, the book produces "no new concepts of medicine or surgery" but it does contain a great deal of information that will be very useful to those for whom it is written. The one thing that this book demonstrates most clearly is that the department organised to treat "what nobody else wants" should no longer have a place in the well organised hospital service. If the general practitioner service is working properly it seems unnecessary to have a hospital department devoted to the "casual" patient. It is to be hoped that the name, which still means exactly what the author says in many hospitals, will soon begin to disappear.—J. C. Scott.

ESSENTIALS OF ORTHOPAEDICS. By Philip Wiles, M.S., F.R.C.S., F.A.C.S., Senior Orthopaedic Surgeon, Middlesex Hospital, London, and King Edward Memorial Hospital, Ealing; Lecturer in Orthopaedic Surgery, University of London; President Elect, British Orthopaedic Association. Second edition. 10 × 7 in. Pp. xv + 538, with 393 figures and 7 colour plates. Index. 1955. London: J. & A. Churchill Ltd. Price 55s.

The second edition of Mr Wiles's text-book is extremely welcome, especially to those who have had practical experience of the help it has accorded to students before and after graduation, and to surgical residents. The expansion of the book is moderate in comparison with the first edition, fifty pages having been added by wise revision of all sections, particularly those on back pain and sciatica, congenital dislocation of the hip, osteoarthritis of hip and knee, ischaemic contractures and osteomalacia. The section on the shoulder girdle remains outstanding in its clarity and excellence of presentation, and the number of textual illustrations has been increased both in number and quality. If it were possible to frame synoptically a general section on the basic principles of fracture treatment, Mr Wiles will be the person to do it, and perhaps further editions, which will without doubt be called for, may contain such a section. The firm reputation which this work has so deservedly gained amongst students and residents during the last five years makes the addition of a bibliography section also doubly desirable. In a work such as this, where wisdom and commonsense have winnowed the tares from the wheat, the interested student would benefit greatly from a readily accessible list of the classical references.—Harry Platt.

LES ARTHROPLASTIES DE LA HANCHE. By Jean Debeyre, Professeur agrégé à la Faculté de Médecine de Paris, Chirurgien des Hôpitaux; and Paul Doliveux, Aide d'Anatomie et Chef de clinique chirurgicale à la Faculté de Médecine de Paris. 9¾ × 6¼ in. Pp. 91, with 12 figures. 1955. Paris: Editions Médicales Flammarion. Price 1,700 fr.

This monograph is based on the experience of the authors in operating on 400 hip joints. The detailed follow-up on 178 arthroplasties of a minimum duration of two years and a maximum

of five years is included. The first part of the book is concerned with the applied anatomy and physiology of the hip joint. The mechanics are summarised very lucidly and well, and the advantages and the defects of the various types of prosthesis are carefully examined. The prostheses employed were mainly acrylic of the Judet type, but Smith-Petersen vitallium cups and a few acrylic prostheses of the type favoured by Merle D'Aubigné were also used. But as the authors point out, theory and practice do not always accord, particularly in the case of arthroplasty, whose unpredictability is one of its great disadvantages.

A valuable chapter is devoted to causes of failure. It is not always possible to determine why an arthroplasty has failed. Certainly one of the main problems is in the neck of the femur; the authors show conclusively that, whatever the angle or length of the neck, it can never achieve perfect mechanical equilibrium and will always be a site of weakness that will throw strain on a prosthesis. Absorption of the neck, the reasons for which are not always clear, is another cause of trouble. The authors conclude that the original Judet prosthesis is far from satisfactory, for mechanical reasons, and they have abandoned it. They are doubtful if metal ones of the same type will be very much better. They prefer the new prosthesis of Merle D'Aubigné; but they are a little concerned about the sacrifice of so much bone for its insertion, and rather hesitate to use it. They believe it to be of great value in the failed case, for which they also perform the Girdlestone excision of the head and neck of the femur.

The results in general are those noted elsewhere: about one-third good and one-quarter poor. The operation for arthroplasty should never be advised in a young patient, and even in other patients should be very carefully considered beforehand.

This little book should be read by all who are interested in hip surgery. It confirms most of what we know and suspected about arthroplasty. It is clear that the biomechanical problem of the hip joint is a difficult one to solve, and the reader will be left in no doubt that the ideal arthroplasty is still beyond our reach. The union between living tissue and an inert substance will always be a point of weakness.—F. C. Durbin.

RECENT ADVANCES IN THE STUDY OF THE STRUCTURE, COMPOSITION, AND GROWTH OF MINERALIZED TISSUES. Annals of the New York Academy of Sciences. Volume 60, Art. 5. Edited by Roy Waldo Miner. Consulting Editor Roy O. Greep, with 19 contributors. 9 × 6 in. Pp. 265, with many figures and tables. 1955. New York: The Academy. Price 28s.

BONE: An Introduction to the Physiology of Skeletal Tissue. By Franklin C. McLean, Ph.D., M.D., Emeritus Professor of Pathological Physiology, University of Chicago; and Marshall R. Urist, M.D., Associate Professor of Surgery (Orthopaedics), University of California. 9 × 5½ in. Pp. xii + 182, with 26 figures. Index. 1955. Chicago: The University of Chicago Press. London: Cambridge University Press. Price 45s.

BONE AND BONES. Fundamentals of Bone Biology. By Joseph P. Weinmann, M.D., College of Dentistry, University of Illinois; and Harry Sicher, M.D., D.Sc., School of Dentistry, Loyola University, Chicago. Second edition. 10 × 7 in. Pp. 508, with 302 figures. 1955. London: Henry Kimpton. Price £5.

Knowledge of the intimate structure and chemistry of bone has advanced far beyond the days when it was the hobby of a few back-room boys. It is reaching the stage when it is of practical importance in the daily work of all concerned with the treatment of diseases and injuries of bones. The time has come when every orthopaedic surgeon who wants to be more than a carpenter should realise that he must add chemistry and physics to his present skill as a surgeon, an engineer and a plasterer. Three recently published volumes will keep him abreast of modern developments.

The report of the conference held at the New York Academy of Sciences in January 1954 is published in its Annals of April 1955. A wide field is covered but the contributions deal particularly with the structure and chemical composition of bone, dentine and enamel, as studied with the aid of the electron microscope, radiotracers and x-rays; there are also valuable studies on nutritional and hormonal influences.

McLean and Urist deal with the same subject; but the material is presented in the form of a monograph which surveys the whole field in a systematic manner, and, in the final chapters, they endeavour to relate the new knowledge to old problems such as the healing of fractures. Their views on the histogenesis of bone and its chemical composition are very clearly expressed and can be readily understood by anyone who has had a basic scientific education. The book should be studied by every orthopaedic surgeon.

Bone and Bones by Weinmann and Sicher is the second edition of an already well known book. In the first part bone is studied as a tissue, and its normal growth and structure are described; the second part deals with the pathology of bone and covers every disorder of the skeleton—developmental, structural, hormonal, inflammatory and neoplastic. The first edition was welcomed as a refreshing attempt to co-ordinate the complex rivalries of the clinical, pathological and experimental points of view. The second edition, although published eight years later, contains very few changes. This is a little disappointing in view of the advances that have been made in nearly every field.—Philip Wiles.

MODERN TRENDS IN BLOOD DISEASES. Edited by John F. Wilkinson, M.D., M.Sc., Ph.D., F.R.C.P., F.R.I.C. Consultant Physician, United Manchester Hospitals, Director, Department of Haematology, Manchester Royal Infirmary. 9¾ × 7 in. Pp. viii + 359, with 91 figures, 2 colour plates and many tables. Index. 1955. London: Butterworth & Co. (Publishers) Ltd. Price 65s.

This work is a reminder of the wide expanse of general medicine and of its ramifications that is touched by orthopaedics. Indeed, the very first chapter deals with "Bone Changes and Blood Diseases" in a masterly survey by D. Lloyd Griffiths.

Blood diseases make their mark on bone largely, but not exclusively, through the marrow, but not all conditions affecting the marrow can be called blood diseases, vague though this name is. Consequently such important conditions as the lipoidoses have had to be excluded; but Griffiths has found good reasons for including Hodgkin's disease, and he has dealt critically with non-leukaemic diseases of the bone marrow, notably reticulum-cell tumours, lymphosarcoma, myelomatosis and conditions called "Ewing's tumour."

The blood diseases coming first to mind are the leukaemias—especially in their aleukaemic stage—with their destructive, proliferative and ischaemic changes in bone. Lymphatic leukaemia is much the commonest to give bone changes, especially in the young, and Griffiths believes that the growing skeleton is demonstrably affected in all cases of lymphatic leukaemia except the most fulminating. On the other hand bone changes are said to be very uncommon in chronic leukaemia of adults, but the reviewer believes that misdiagnosis considerably masks their true incidence. The importance of pain as the outstanding symptom of leukaemic bone affection is stressed, and especially the danger of ascribing the pain to some trivial condition in childhood or, if joints are involved, to acute or subacute rheumatism.

Reference is made to some of the less familiar blood diseases—Cooley's erythroblastic anaemia, myelosclerosis with and without osteopetrosis, the spherocytic anaemia of familial haemolytic jaundice, and sickle-cell anaemia, in which the occasional resemblance of the hip to that of Perthes' disease might merit mention.

This volume is characterised by the poor radiographic reproduction that was criticised so severely in our review of the companion volume, *Modern Trends in Orthopaedics* (Journal of Bone and Joint Surgery, 1950, **32-B**, 444). We would condone the occasional poor figure included to oblige an author with a poor radiograph of a unique or specially interesting condition, but that is another matter.—H. Jackson Burrows.

PELVO-SPONDYLITIS OSSIFICANS. Rheumatoid or ankylosing spondylitis. By Ragnar Romanus and Sven Ydén. Karolinska Sjukhuset and King Gustaf V's Research Institute, Stockholm. 11 × 8¼ in. Pp. 161, with 55 figures. Index. 1955. Copenhagen: Ejnar Munksgaard. Price Dan. kr. 45.

Dr Romanus in 1953 published a thesis on the connection between ankylosing spondylitis and chronic urogenital infections in men, especially seminal vesiculitis. His interest in the manifestations of ankylosing spondylitis has led to the volume under review which is jointly written by Dr Romanus, Associate Professor of General Surgery and Chief of the Surgical Out-patient Department, Karolinska sjukhuset, Stockholm, and Dr Sven Ydén who is Roentgenologist at Karolinska sjukhuset. The book is divided into two sections; the first an unillustrated text covering about seventy pages, and the second of about equal size devoted to excellently reproduced radiographs illustrating the radiological manifestations of the disease in the spine and elsewhere.

The authors have paid particular attention to the early changes to be seen in the spine which are less familiar to most doctors than the well known picture of the final ankylosing stages. These early radiological changes are considered in very great detail and well illustrated by successive radiographs showing the development of the changes. In the unillustrated text the clinical and

radiological manifestations are considered in great detail. Passing reference is made to possible causes of the condition which includes the statement that " chronic non-specific urogenital infection was demonstrated in practically every case (prostato-vesiculitis, often utriculitis, Cowperitis)." No indication is given of the evidence upon which this statement is based, and, although the authors speak of the importance of detecting such infections early, they make no claim that early detection and treatment have any influence in arresting or diminishing the disease. Assuming chronic urogenital infection to be the cause, the spread of infection is said to follow the same pattern as that of prostatic secondary carcinoma via the vertebral venous system.

The chief value of the book is as a reference work in which the early radiographic signs of ankylosing spondylitis are described and illustrated. It does not attempt to deal with the orthopaedic and other treatment of the condition. It would not appeal to the ordinary orthopaedic reader. In reading it, it is sometimes irritating to have to turn from the unillustrated text to the atlas at the end of the book to follow the points raised in the text, but this is, perhaps, unavoidable.—Norman ROBERTS.

THE MANAGEMENT OF ACUTE POLIOMYELITIS. By C. P. STOTT, S.R.N., Diploma of Poliomyelitis Nursing (U.S.A.); and M. FISCHER-WILLIAMS, M.R.C.P. (Ed.), Senior Neurological Registrar, Birmingham United Hospitals, Formerly Neurological Registrar, Oxford United Hospitals. Foreword by W. Ritchie RUSSELL, C.B.E., M.D., F.R.C.P. (Lond. and Ed.), Consultant Neurologist, United Oxford Hospitals. $8\frac{3}{4} \times 5\frac{3}{4}$ in. Pp. xii + 99, with 43 figures. Index. 1955. Edinburgh and London: E. & S. Livingstone Ltd. Price 12s. 6d.

In this book the authors have achieved what they set out to do. In fourteen chapters all aspects of the acute disease are covered, including isolation technique, the physiology of respiration, and the nursing care of patients in respirators. There is continual emphasis on the need for gaining

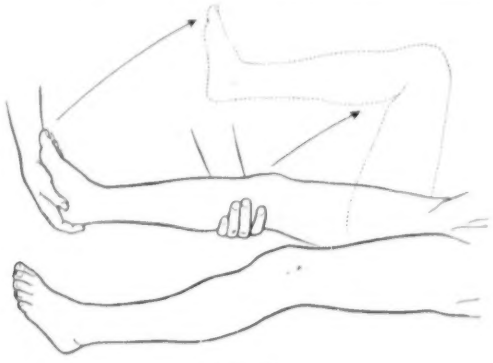

FIG. 38
Hip and knee flexion.

the patient's confidence and of providing a sense of security. The importance of never leaving alone a patient who is in a respirator, and the denunciation of trickery when weaning from the respirator, are illustrative of this theme. In the early days the responsibility for posturing paralysed limbs, and for carrying out such passive movements of joints as are necessary, must fall to a great extent upon the nursing staff; the diagrams illustrating such treatment are very informative. With its first class production this is one of the best books of its kind that have appeared in recent years. It will prove very helpful to those doctors and nurses whose work brings them in contact with the early case of poliomyelitis.—Donal M. BROOKS.

SERVICES FOR THE DISABLED. Standing Committee on the Rehabilitation and Resettlement of Disabled Persons. $9\frac{1}{2} \times 6$ in. Pp. v + 88, with 25 figures. Index. 1955. London: Published for the Ministry of Labour and National Service by Her Majesty's Stationery Office. Paper cover. Price 4s. 6d.

Clearly this country is unique in providing such comprehensive facilities for the rehabilitation of the disabled. For the first time " a simple, concise and factual account of the provision now made

in the United Kingdom for the rehabilitation and resettlement of the disabled " is available in one volume. This book is a mine of information and will prove a valuable source of reference for all doctors.

When the pages are turned over it might appear that this problem has virtually been solved, but rehabilitation depends for its success upon the full co-operation of doctors, employers, rehabilitation centres, disablement resettlement officers (D.R.O.s) and many others. With this complicated machinery there is the constant danger that the patient may lose his way in the maze of multiple responsibility for his future resettlement. To whom should he turn for advice? The D.R.O., the Almoner, his general practitioner, or his surgeon? To counteract this difficulty it is essential that the services in any one hospital should be integrated, and the establishment of resettlement clinics has proved very helpful. The responsibilities of the hospital should not cease once the patient has been referred to the appropriate D.R.O. A regular system of follow-up after resettlement is just as important as its medical counterpart. In many instances the D.R.O. has not the time or the appropriate facilities to seek out work for the many patients referred to him. For this reason there is much to be said for the advent of the Hospital Resettlement Officer who can maintain a liaison between the hospital, the patient and the employer. More than ever nowadays it becomes apparent that personal contact is of the greatest value: this sort of work cannot be carried out adequately from an office.

The special problem of the young chronic sick has yet to be resolved. This particularly applies to those severely paralysed by poliomyelitis, often dependent upon a respirator for the rest of their lives. These patients frequently come from homes to which they can never return, either because accommodation is unsuitable, or because there is no one capable of looking after them. They are not sufficiently fit to qualify for a residential rehabilitation centre; and they therefore linger on in orthopaedic hospitals, blocking beds that could be more suitably employed. Tucking them away in institutions is not the answer to this problem. Rehabilitation in its fullest sense must mean the preservation of the family circle, if at all possible. It may well be that a foundation on the lines of the Papworth Village Settlement will prove to be the solution.—Donal M. Brooks.

COMMENT TRAITER LES VARICES ET LEURS COMPLICATIONS. By Raoul Tubiana, Chirurgien de l'Hôpital Américain de Paris, Ancien Chef de Clinique à la Faculté de Médecine. 8¼ × 6 in. Pp. 160, with 5 figures. 1955. Paris: Editions Médicales Flammarion. Price 550 fr.

The author begins by telling us that in the United States it is estimated that there are one and three-quarter million people with varicose veins and it is not difficult to believe that the proportion of Western Europeans so afflicted is equally high. No surgeon can ignore this miserable complaint and all the ills resulting from it. This book, written by a most able orthopaedic surgeon, who is becoming well known in this country, is one of a series of therapeutic handbooks which manage to combine sound advice with quite astonishing brevity. Tubiana knows his subject and writes about it with the easy grace of an authority. Although it is a popular rather than an academic work—with plenty of italics to emphasise important points—it is a thoroughly serious production and will be warmly welcomed by those interested in the treatment of varicose veins.—H. J. Seddon.

BOOKS RECEIVED

These books are acknowledged with grateful thanks. Selections will be made for review as space permits and in accordance with their interest for our readers.

SYMPOSIUM ON FATIGUE. Edited by W. F. Floyd and A. T. Welford. The Ergonomics Research Society. Proceedings Volume I. 1953. London: H. K. Lewis & Co. Ltd.

SYMPOSIUM ON HUMAN FACTORS IN EQUIPMENT DESIGN. Edited by W. F. Floyd and A. T. Welford. The Ergonomics Research Society. Proceedings Volume II. 1954. London: H. K. Lewis & Co. Ltd.

CEREBRAL PALSY. By Cyril B. Courville, M.D. 1954. Los Angeles: San Lucas Press.

PERIPHERAL NERVE INJURIES. Edited by H. J. Seddon, C.M.G., D.M., F.R.C.S. Medical Research Council Special Report Series No. 282. 1954. London: Her Majesty's Stationery Office.

CHIRURGIE RÉPARATRICE. By R. Merle D'Aubigné and twelve other contributors. 2me Serie. 1955. Paris: Expansion Scientifique Française.

FRACTURES IN CHILDREN. By Walter P. Blount, A.B., M.D., F.A.C.S. 1955. London: Baillière, Tindall and Cox.

NEURO-VASCULAR HILA OF LIMB MUSCLES. By James Couper Brash, M.C., M.A., M.D., D.Sc., LL.D., F.R.C.S. (Ed.), F.R.S.E. 1955. Edinburgh and London: E. & S. Livingstone Ltd.

RECENT ADVANCES IN RADIOLOGY. By Thomas Lodge, M.B., Ch.B. (Sheff.), F.F.R., D.M.R. Third edition. 1955. London: J. & A. Churchill Ltd.

BONE AND JOINT X-RAY DIAGNOSIS. By Max Ritvo, M.D. 1955. London: Henry Kimpton.

THE STRUCTURE, COMPOSITION AND GROWTH OF BONE, 1930–1953. A Bibliography compiled by Marjory C. Spencer and Katherine Uhler. 1955. Washington: Superintendent of Documents U.S. Government Printing Office.

RADIOGRAPHIC ATLAS OF SKELETAL DEVELOPMENT OF THE KNEE. A Standard of Reference. By S. Idell Pyle, Ph.D., and Normand L. Hoerr, Ph.D., M.D. 1955. Springfield, Illinois: Charles C. Thomas, Publisher. Oxford: Blackwell Scientific Publications.

BODY FLUIDS IN SURGERY. By A. W. Wilkinson, Ch.M., F.R.C.S.E. 1955. Edinburgh and London: E. & S. Livingstone Ltd.

THE SPINE. A Radiological Text and Atlas. By Bernard S. Epstein, M.D. 1955. London: Henry Kimpton.

THE LIFE OF LORD NUFFIELD. A Study in Enterprise and Benevolence. By P. W. S. Andrews and Elizabeth Brunner. 1955. Oxford: Basil Blackwell.

LOCAL ANALGESIA HEAD AND NECK. By Sir Robert Macintosh, D.M., F.R.C.S.E., F.F.A.R.C.S., and Mary Ostlere, M.B., M.R.C.P.E., F.F.A.R.C.S. 1955. Edinburgh and London: E. & S. Livingstone Ltd.

ATLAS OF RUSH PIN TECHNICS. A System of Fracture Treatment. By Leslie V. Rush, M.D., F.A.C.S., F.I.C.S. 1955. Meridian, Mississippi: The Berivon Company.

TRAITÉ DE TECHNIQUE CHIRURGICALE. Généralités, Chirurgie des Tissus, Chirurgie des Membres. By S. Oberlin. Volume I. Second edition. 1955. Paris: Masson et Cie.

FRACTURES OF THE FACIAL SKELETON. By N. L. Rowe, F.D.S.R.C.S., L.R.C.P., M.R.C.S., L.M.S.S.A., H.D.D.R.C.S., and H. C. Killey, F.D.S.R.C.S., L.R.C.P., M.R.C.S., L.M.S.S.A., H.D.D.R.C.S. 1955. Edinburgh and London: E. & S. Livingstone Ltd.

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We acknowledge with thanks receipt of:

Acta Chirurgica Scandinavica, and Supplements
Acta Ortopedica-Traumatologica Iberica
Annals of the Royal College of Surgeons of England
Archives of Surgery
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British Journal of Surgery
British Medical Bulletin
Journal of Neurology, Neurosurgery and Psychiatry
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THE INDEX

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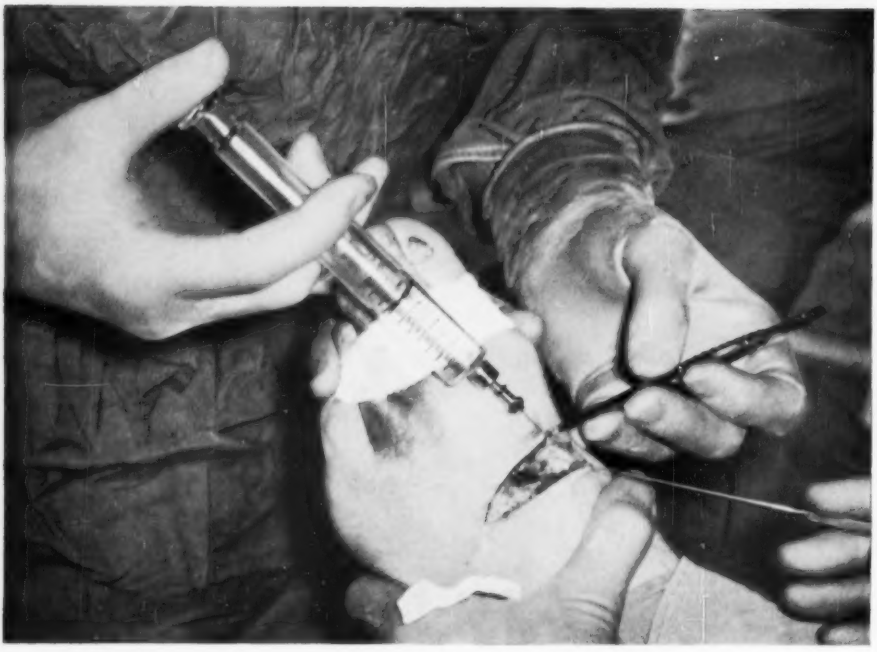

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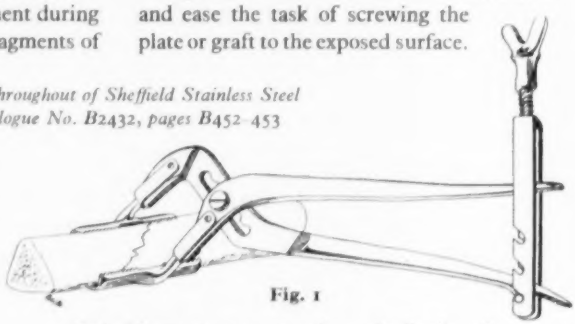

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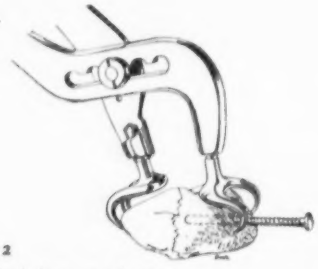

Fig. 2
Hooked jaws used to grip a fractured patella.

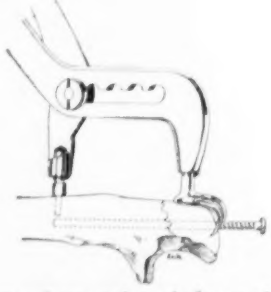

Fig. 3
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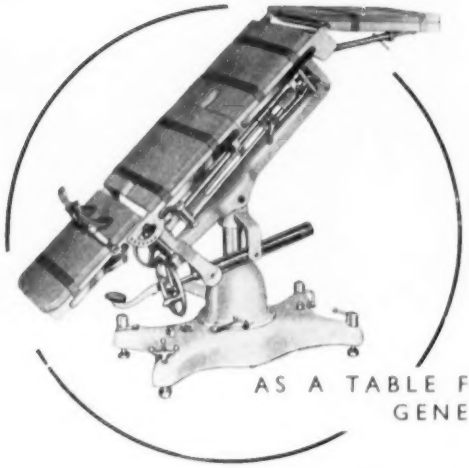

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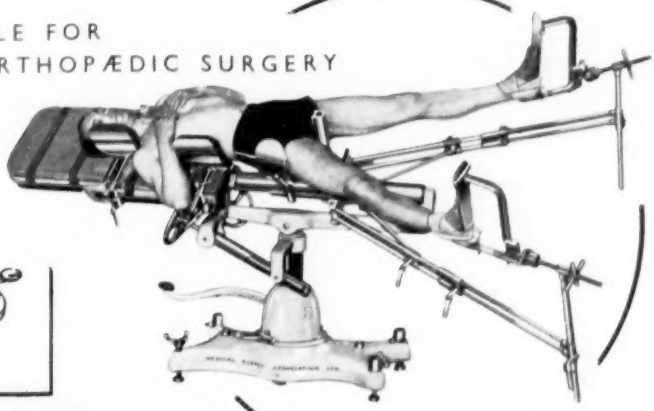

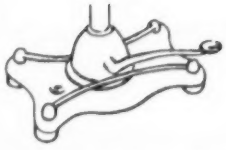

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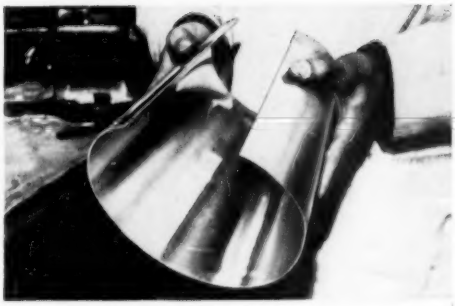

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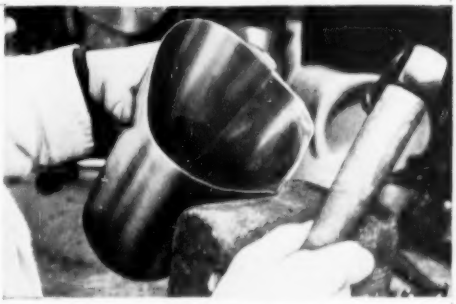
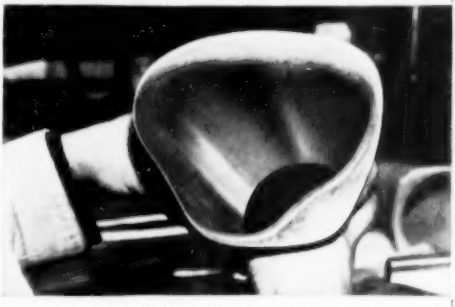


Preparing to shape anatomical features of socket.



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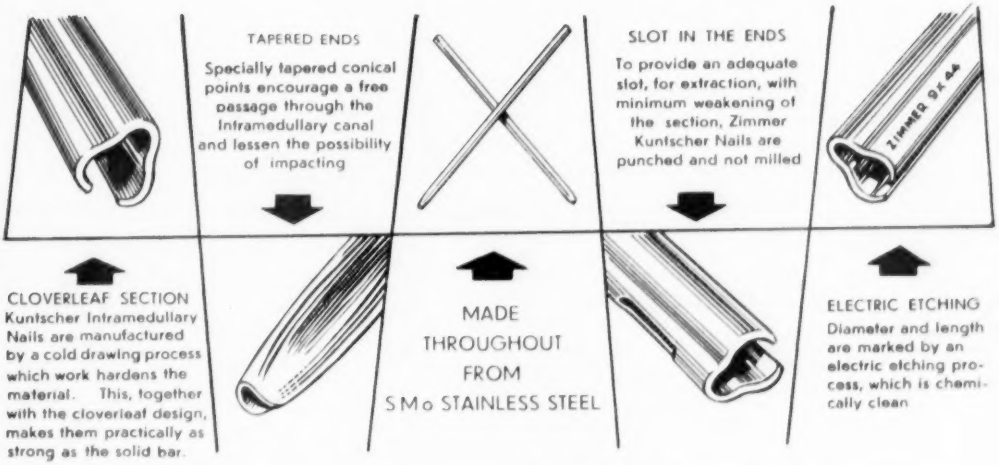
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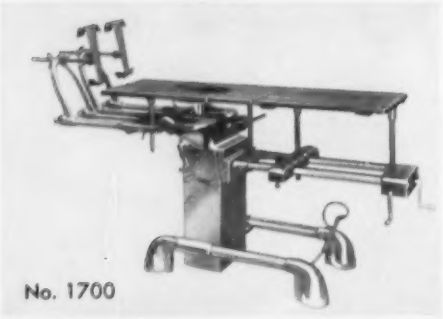

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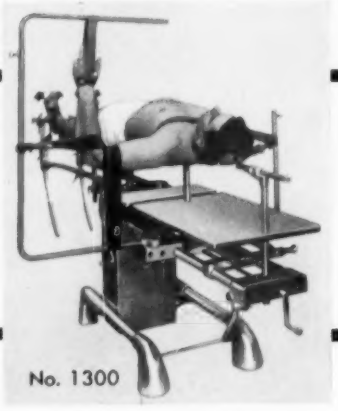

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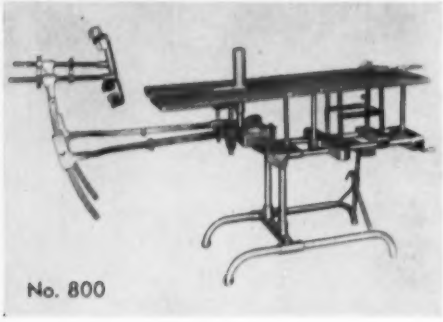

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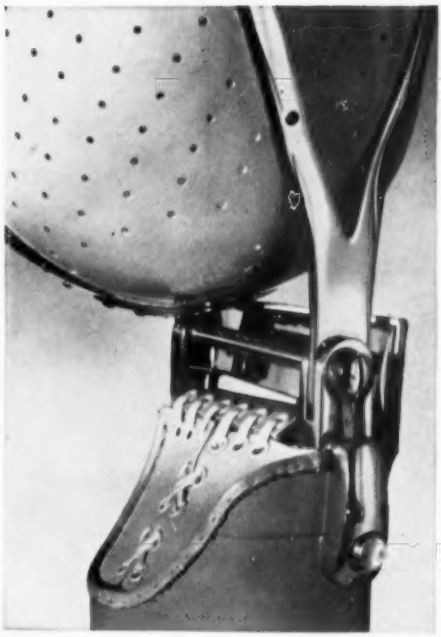

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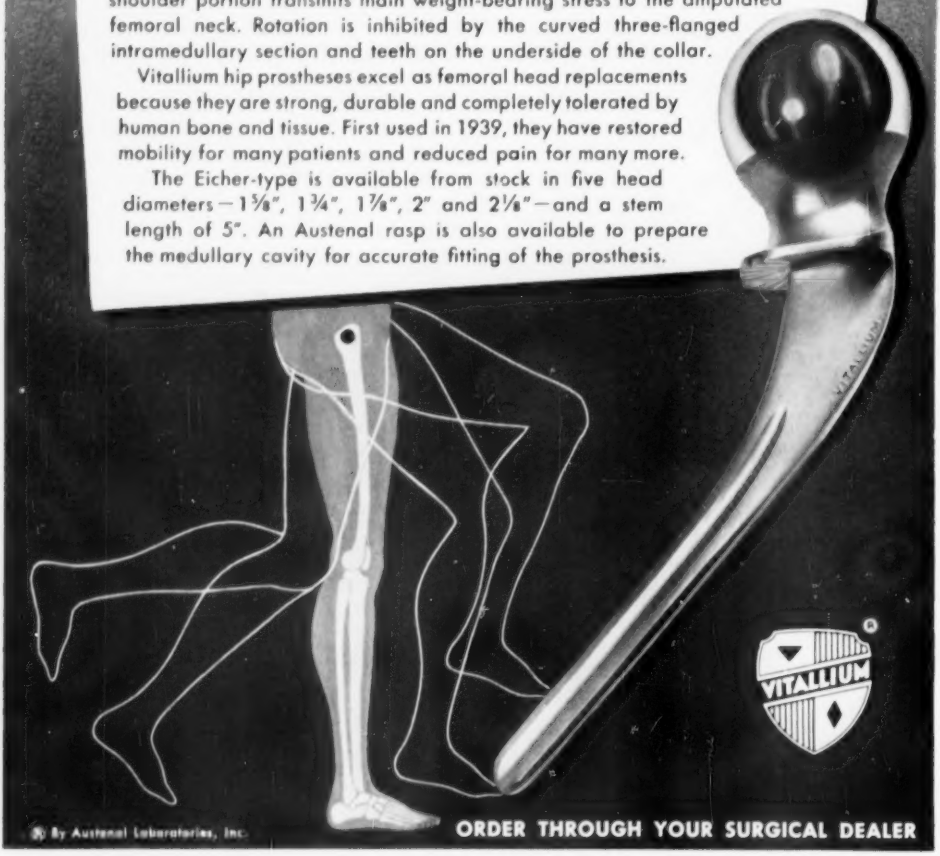

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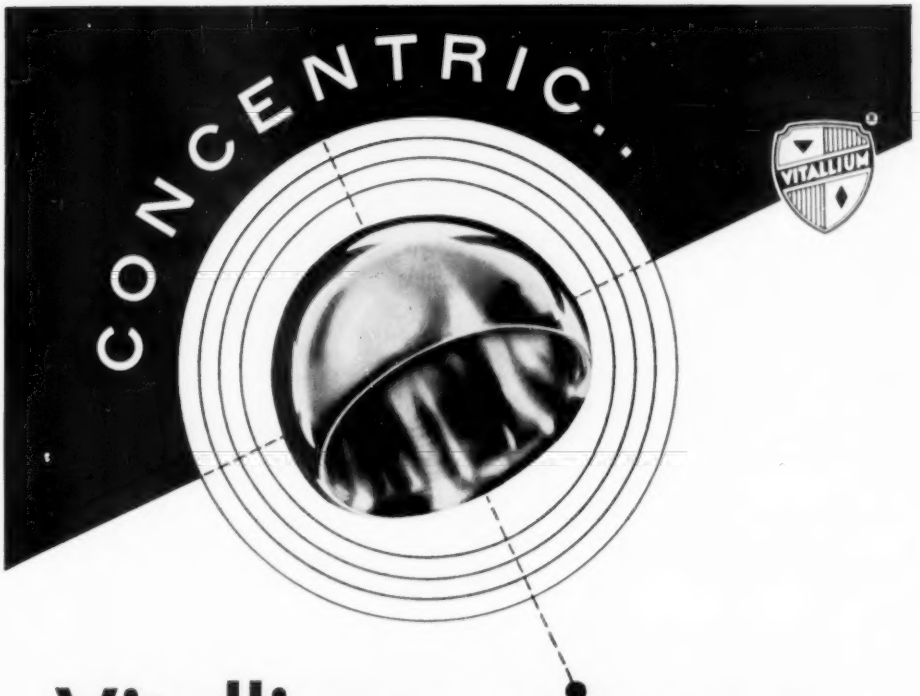

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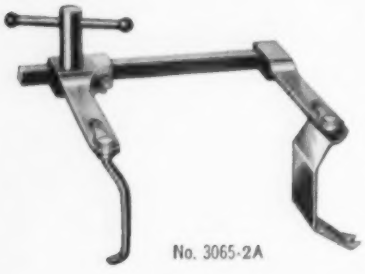

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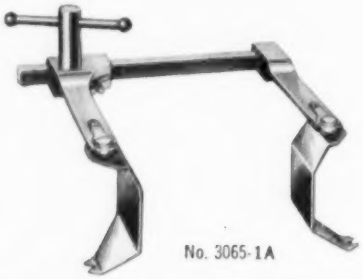

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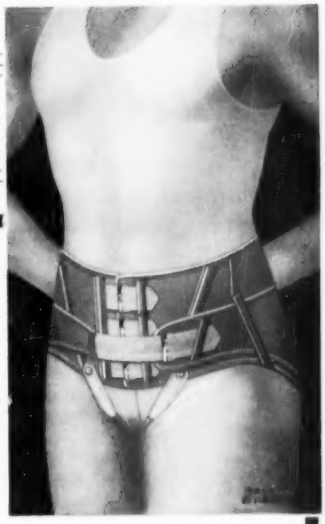

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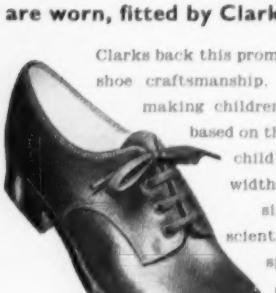

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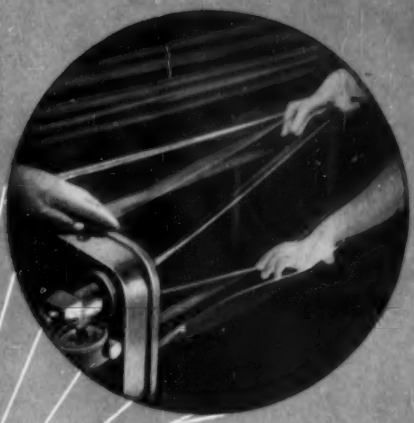

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